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PEMPHIGUS OF THE CONJUNCTIVA.

CAPTAIN RAY CONNOR, M. D., M. R. C., AND CAPTAIN CHARLES A. BURKHOLDER,

M. D., M. R. C.

U. S. A. GENERAL HOSPITAL, NO. 11, CAPE MAY, N. J.

Report of an atypical case having a history of eight years duration, with colored plate showing lesions on the eyeball.

The opportunity having presented itself to us, to have under our observation for a number of weeks an atypical case of pemphigus of the conjunctiva, it seemed worth while to put it on record, especially in view of the paucity of illustrations of this condition. In some particulars our case followed very closely the classical descriptions of the disease, but it lacked the general symptoms and affected one eye only. This condition is so uncommon that Casey A. Wood has reported that he has seen but three cases in twenty-five years of his practice.

Pemphigus is an acute or chronic disease of the skin and mucous membranes, characterized by the formation of rounded or oval blebs, arising from apparently normal surfaces, which may or may not be accompanied by constitutional symptoms. Chronic pemphigus runs a long course, the bullae continuing to appear and reappear either singly or in crops. Frequently the mouth and throat may exhibit similar blebs, and though rarely, the conjunctiva may be attacked.

Pemphigus has been known to arise primarily in the conjunctiva, and only later involve the skin, although the contrary order is the usual sequence. General pemphigus is more common in children, but ocular pemphigus is an affection of adult life. Statistics show the average age is about 41 years. The

bullae are rarely seen on mucous membranes, because, probably, of the delicacy of their structure. The conjunctiva exhibits on examination grey, necrotic areas or patches deprived of epithelium and covered with grey lardaceous secretion. Inflammatory reaction following the bullae soon penetrates the subconjunctival tissues, and is accompanied by the development of new blood vessels which lead to the formation of cicatrices. Those in turn lead to excessive contraction and shrinking of the conjunctiva, and symblepharon.

C. L. E., a private, 25 years old, was admitted to the U. S. A. General Hospital No. 11, and assigned to the service of Major Burton Chance, April 1, 1918. The patient complained of an affection of his left eye, which has incapacitated him from military duty for the past nine months. His past and family history were unimportant. He admitted gonorrhea 6 years ago, with a previous soft chancre. He had been in the service over four years and able to do full duty until July 8, 1917. He however gives a history of recurrent attacks of inflammation of the left eye for the past 7 or 8 years, which would last for three or four days and then subside. Exacerbations would sometimes come on every month, and at other times the eye would remain clear for several months.

No particular attention was paid to these attacks and the patient was sent to France with the early expeditionary forces. While in France, owing to his increased activities, the attacks became of such a nature that he was compelled to report to the hospital for treatment early in July. Lieutenant Lloyd B. Whitham, M. D., M. R. C., had him under observation in Base Hospital No. 18 for about three months and noted the bullae constantly recurring, either singly or in pairs, both on the bulbar and palpebral conjunctiva. These lasted but a short while and left a greyish membrane. The conjunctiva gradually atrophied and caused a perceptible loss of the inferior conjunctival fornix. He made a diagnosis of pemphigus of the conjunctiva, which was not concurred in by Major Wile, the Professor of Dermatology at the University of Michigan. Major Wile saw the case several times and preferred to call it a pemphigoid affair, thinking it was an exudative erythema.

On admission April 1, 1918, the following conditions were noted:

Vision, R. = 20/30; L. = 20/100.

The left eye showed a slight photophobia, and considerable mucopurulent discharge. Conjunctiva showed marked brownish stain or discoloration on eyeball and in lower culdesac (probably from silver treatment). Lower fornix was almost obliterated by adhesive bands. The left cornea had a linear cicatrix in lower nasal quadrant near limbus, a haziness in outer quadrant and an old fine cicatrix extending across limbus (all evidently results of earlier ulcerations). Iris was clear and reacted well to light and accommodation. Lens and other media clear. Fundus normal. No degree of hyperopia present. The right eye was entirely normal and showed no trace of previous inflammation.

On May 14th, 1918, he showed the first distinct blebs noted while under our care. There were two small blebs, as shown in the accompanying illustration (Pl. XI, Fig. 1), about 1 mm. in diameter, on the bulbar conjunctiva just below limbus. These showed a

sloughing greyish yellow base, slightly raised and surrounded by a local area of conjunctival injection. These areas appeared in less than 24 hours and gradually disappeared in about 3 days. In succeeding weeks, several small lesions of a similar character appeared on the ocular conjunctiva, only to disappear in a few hours.

On June 5th, 1918, a large irregular sloughing area was noted on the ocular conjunctiva 5 mm. external to the limbus in a horizontal line (Pl. XI, Fig. 2). This patch was somewhat raised, 4 mm. in diameter, with a dirty greyish yellow sloughing surface. A small area of conjunctival injection surrounded it. This lesion also disappeared in a few days. No lesions were noted on the palpebral conjunctiva during the period of our observations.

Smears from the conjunctiva taken a number of times showed pus cells, but no bacteria of pathologic significance.

A blood examination showed:

Hemoglobin	99%
Red blood corpuscles.....	5,912,000
White blood corpuscles	8,400

Differential blood count:

Small mononuclears	24%
Large mononuclears	4%
Transitionals	4%
Polymorphonuclear neutrophiles	66%
Polymorphonuclear basophiles	2%

Wassermann negative.

General physical examination negative.

On his discharge from the army, June 14th, 1918, the eye was comparatively quiet and there were no active lesions evident. The conjunctiva showed a dry, brownish, lustreless surface, and the shrinking of the membranes was much in evidence in the lower culdesac, where broad adhesive bands connected the palpebral and bulbar conjunctiva.

From a close study of the lesion and perusal of the available literature, we have come to the conclusion that this was a true case of pemphigus of the conjunctiva. The lesion itself was identical with those described by other observers. It developed the same way,

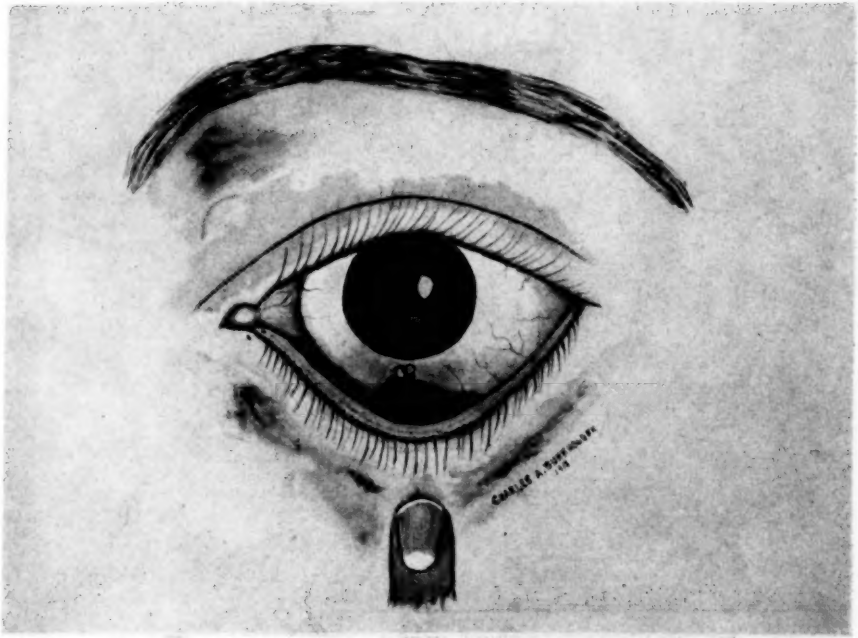
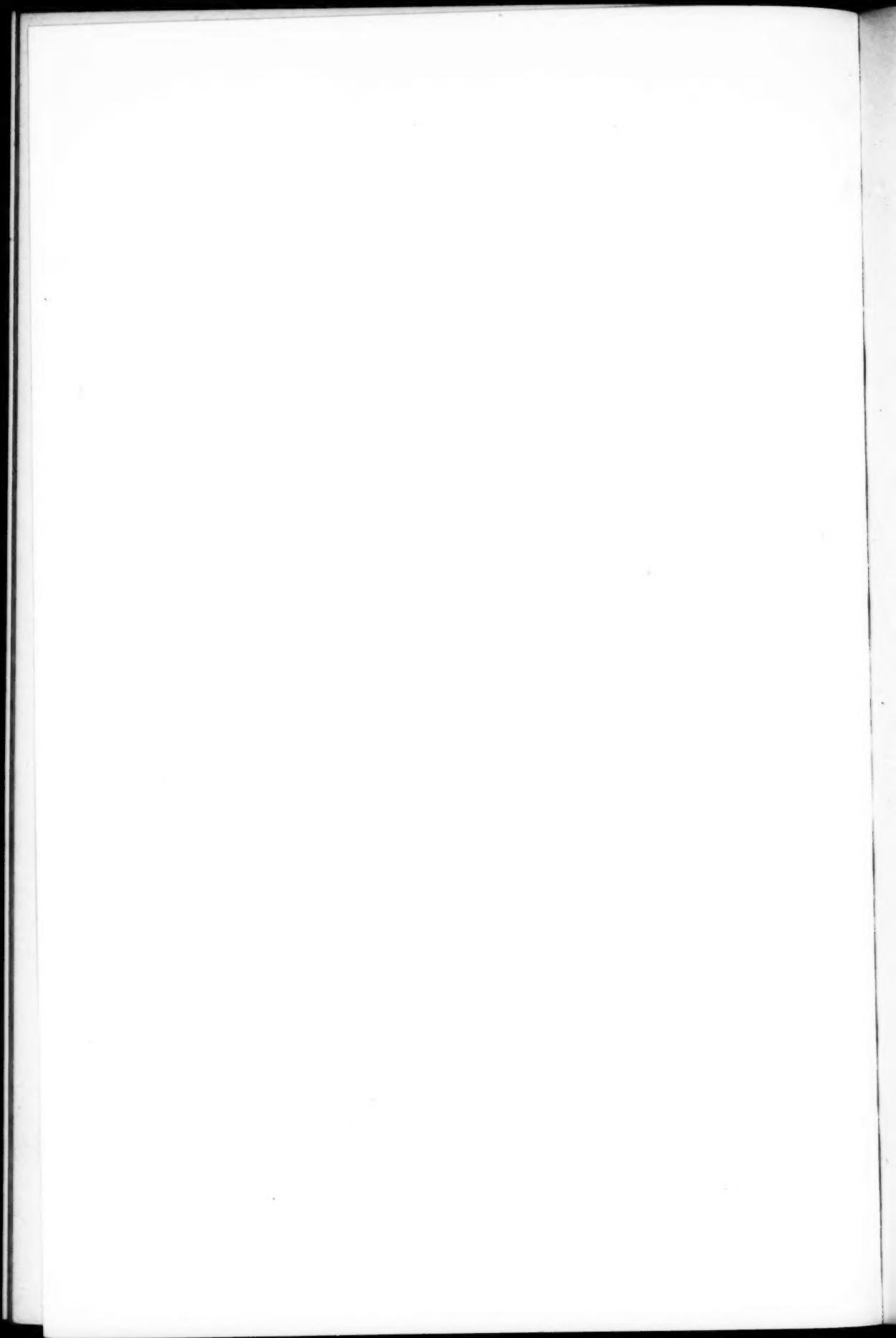


FIG. 1. PEMPHIGUS OF CONJUNCTIVA



FIG. 2. PEMPHIGUS OF CONJUNCTIVA
CASE OF CONNOR AND BURKHOLDER



ran the same course and left identical sequellae. It occurred as usual in an adult and ran a very stubborn and intractable course. The associated lesions of general pemphigus, or lesions in the mouth and throat were, however, lacking as were lesions in the other eye. There is, however, no certainty that the immunity of the other eye will continue, or that general

symptoms may not supervene at any time. At present it must be classified as a mild form of the disease, with a very persistent and chronic course.

Our thanks are due to Major Burton Chance for his kindness and assistance in working up this case, and from whose article on pemphigus in the "American Encyclopedia of Ophthalmology" we have freely drawn.

QUININ POISONING, ITS OCULAR LESIONS AND VISUAL DISTURBANCES.

LT.-COL. R. H. ELLIOT, I. M. S., RETIRED.

LONDON, ENGLAND.

This analytic review of the literature of the subject combined with the personal experience and conclusions of its author constitutes a clear statement of our present knowledge of impairment of vision by quinin poisoning. It is of especial value at this time.

A titanic world war has flung masses of young men of the Anglo-Saxon race into strange parts of the earth, where they are exposed to the attacks of malaria and other fevers, in a manner hitherto quite unprecedented. The result has been that a large number of our medical men have been comparatively suddenly forced to take a practical interest in diseases, of which they previously knew but little. Their response to the call of duty and of science has been magnificent, but it would be strange if the position were not beset with danger. At a recent discussion on the treatment of malaria before the Society of Tropical Medicine (London, March 15th, 1918), tropical experts of the first calibre discussed the doses of quinin, which had been advocated on the strength of reports received from military surgeons; and the writer sat astonished to hear of one medical man giving 90 grains a day for 7 days, and further doses thereafter; and of others advocating the use of the drug in quantities which appeared enormous. The problem that today interests the surgeons of the British Empire, and the young men un-

der their charge, will shortly enfold the gallant armies, which are crossing the Atlantic Ocean to take their stand by our side in the great struggle for the liberty of the world.

The writer has recently read very carefully the whole literature of quinin poisoning, and has been profoundly impressed by the great possibilities for harm that may lie concealed in the present movement towards unrestricted use of the drug. It is 22 years since de Schweinitz dealt with quinin poisoning in his classic work on toxic amblyopias; and tho, both before and since that time, a very large literature has grown round the subject, no effort appears to have been made to deal with it at all exhaustively, within the limits of a single communication. The present would, therefore, seem to be a suitable time to consider the subject in all its bearings, to review what has been written and learned, and to raise a note of warning, lest in our endeavor to extinguish the germs of malaria, we should be led into the dangers that surround the abuse of quinin. To save a man from death, only to leave him amblyopic, hemeralopic,

and with contracted fields is to render him a poor service indeed; and one for which many would curse and not bless us. The oft-expressed sentiment "I would rather be dead than blind" is one that finds a strong echo in many of our hearts. One purpose of this paper, and not the least important, is to point out how easy it is to diagnose quinin poisoning. The most recently qualified medical man can do it, and he who is armed with this knowledge, will not be led into the danger of mistaking the signs of deep cinchonism for those of malaria, an error, which when committed, is fraught with disaster, and possibly even with death for the patient.

DOSE OF QUININ WHICH MAY GIVE RISE TO EYE SYMPTOMS.

A study of the reported cases of quinin blindness reveals the most startling variations in the dose of the drug required to produce pathologic phenomena in different patients. One will take with impunity a dose which seems simply enormous, whilst another will show failure of vision from a quantity of quinin, which to the ordinary person would be absolutely innocuous. There are, however, certain factors which we must take into account in every case: These are: (1) The salt of quinin employed; (2) the vehicle in which the dose is given; (3) the method of its administration; (4) the total amount of the drug retained in the system; (5) the individual idiosyncrasy of the patient; (6) the age, sex and weight of the patient.

(1) *The salt used.* The sulphat of quinin is probably still employed more commonly than any other preparation, tho the hydrochlorid is very popular. If one may judge from the recent discussion before the Society of Tropical Medicine (London, March 15th, 1918), these two salts stand far ahead of all others in the esteem of experts. The relative solubilities of the various quinin preparations are given below in a tabular form.

Salt of quinin—	Solubility	
	In water	In alcohol
Sulphat	1 in 800.	1 in 65.
Acid sulphat	1 in 10.	1 in 45.
Hydrochlorid	1 in 35.	1 in 3.
Acid hydrochlorid ..	1 in 1.	1 in 5.
Hydrobromid	1 in 55.	1 in 0.7
Salicylat	1 in 630.	1 in 24.

An important observation was recorded at the above meeting of the Society of Tropical Medicine by Dr. Nierenstein, who had analyzed the urines of a number of patients who had been taking quinin salts. He found that the percentage of quinin in the urine was practically constant, regardless of the salt used (sulphat, hydrochlorid or hydrobromid), or of the amounts that had been administered. From this he deduced that there was a limit to the amount of the drug that could be taken up by the blood, and that this limit was indicated by the quinin-content of the urine. He made the suggestion that if a more soluble salt than the sulphat or hydrochlorid were used, the quinin-content of the drug could be greatly raised, thus intensifying the action of the drug on the malarial parasite. The form in which quinin is excreted is in combination with urea. He thought, therefore, that a carbamid of quinin might be the very substance for which tropical physicians were looking. The writer drew attention to the fact that de Schweinitz, in his experiments on animals, had used a carbamid of quinin as well as other preparations, and had found that salt more toxic than any other. Whilst, therefore, it might prove powerfully therapeutic, there was the possibility that by so highly cinchonising the blood, it might act unfavorably on the retina and optic nerve, and determine amblyopia and amaurosis.

It has sometimes been said that quinin is more dangerous than any of the other alkaloids obtained from the cinchona bark. But it seems possible that the true interpretation of this statement lies in the fact that quinin is in much more common use than any of these other substances.

(2) *The vehicle in which the quinin is given.* Some of the patients, who presented pathologic signs, took the quinin in the form of powder, others in pills, others in one form or another of solution; whilst yet others drank comparatively large quantities of alcohol immediately before or afterwards. This must undoubtedly have tended to intensify the action of the drug, by bringing it rapidly into solution in the stomach. On the other hand, the ingestion of quinin in pill or tabloid form introduces a great element of uncertainty into our estimate of the quantity of the drug actually absorbed into the system. Every tropical practitioner knows that stale pills or tabloids of quinin become as hard, and almost as insoluble as stone, and are passed through the alimentary canal from end to end as inert, useless masses. Even when fresh pills have been swallowed, if the quantity is large, the bulk and cohesion of the mass make it easily vomited in the first instance; and disposed to set up irritation, and so be rapidly voided in diarrheal movements, in the second. We shall return to this under our fourth heading.

(3) *The method of administration of the drug.* One of the most interesting points brought out at the recent meeting of the Society of Tropical Medicine was the apparently undivided consensus of opinion that the oral method of administration is far and away the most satisfactory. Not a good word was said for intravenous injection, despite the fact that on the face of it, this would appear to be the only method which attains to absolute certainty in the knowledge of the dose administered. The comments on subcutaneous and intramuscular injection were equally unfavorable. This is not surprising, inasmuch as it has long been known, that when quinin is given in this way, a large quantity of it may remain indefinitely unabsorbed. It has fallen to the lot of many surgeons in the Tropics, to cut down upon and evacuate large masses of the kind, on account of the great irritation the patients suffered from them. Finally, amongst the records of quinin ambly-

opia, one finds a certain number of cases in which the drug was given per rectum, either in the case of children, or of others in whom gastric irritability provoked an intolerance of further administration by the mouth. This is a point to which the most earnest attention of the reader is invited, for it would seem that the little known symptoms of cinchonism were in some of these cases mistaken for those of malaria, and that the drug was consequently pressed, at a time when it should have been totally withheld. It is not difficult to see how disastrous such a course of conduct may prove.

Before leaving this topic, it is of interest to note the various conditions which led to quinin being taken in what proved to be poisonous doses. In the forefront of these, of course, stands administration by medical men, or by the patients themselves, for one or another form of fever, the most common of which was malaria. Many other febrile conditions, however, figure on the list, inclusive of pneumonia, influenza, chills, and numerous ill-described pyrexial states. A mention must also be made of "neuralgias," sometimes considered to be of malarial origin, and of toothache. Next we find a large group of cases in which the drug was deliberately taken for some improper purpose, especially for suicide, or for the production of abortion or miscarriage. Lastly we have the quite considerable group of "accidents." It certainly seems strange that a man or woman should swallow a huge dose of quinin sulphat by mistake for some comparatively innocuous salt, such as cream of tartar, but it unquestionably has been done (Giacomini). In all these self-prescribed instances the drug has been taken by the mouth.

(4) *The total amount of the drug retained in the system.* This point has been taken up several times under the preceding headings. It is obviously one of very great importance in our estimation of "the dangerous dose." In many of the records we learn that the patient vomited quite half the dose given, or that he "passed a large quantity of quinin by the bowel."

Again in the subcutaneous and intramuscular injection, usually no allowance seems to have been made for the fact that much of the drug probably lay inert, where it was first thrown by the syringe. The uncertainties thus introduced into our estimations are too obvious to need emphasizing. When the dose is accurately stated in the first place, it is the outside dose, but, the exact deductions we must make therefrom, in order to arrive at the quantity of the drug that did the damage, are more than open to question. This must be frankly recognized thruout.

(5) *The individual idiosyncrasy of the patient.* From the point of view of the dangers that attend the path of the prescribing physician, when dealing with malaria, this factor probably far outweighs any other. It constitutes the great risk, beside which all others fade into unimportance, and it consequently demands a separate section to itself. For this reason it must be dropped for the present, with the simple comment that in this quinin only falls into line with all the other drugs, which are responsible for producing one or other form of toxic amblyopia.

(6) *The age and sex of the patient.* This probably resolves itself very largely into the weight of the subject, since women are on the average lighter than men, and children than adults. On the other hand, there seems some reason to think that women and children are especially liable to quinin poisoning. Allowance must of course be made for the fact that quite a number of the recorded cases have been in women who desired to produce abortion, and who took large quantities of the drug for that purpose. Probably, too, in some of the attempted suicides the motto was "death or abortion." It is, however, of the utmost importance constantly to keep in mind the dose of quinin in grains, as compared with the total body-weight of the patient. De Schweinitz showed this in his early experiments on animals, and it is so cardinal a rule that it might seem a work of supererogation to insist on it, were it not for the fact that this essential

piece of information is very rarely given in the published records of cases of quinin amblyopia.

When the writer first started collecting the material for the present paper, he did so under the influence of many years of extensive experience with malaria and its treatment in southern India, where his opportunities of meeting with quinin amblyopia would have been exceptional, had such cases really been common. He believes that the dictum of Laveran "not more than 45 grs. of quinin to be given in 24 hours," holds widely there; and to this he attributes the remarkably small number of cases of quinin blindness which he met with in the Madras Presidency. He cannot with certainty remember more than 4 or possibly 5 such cases. Altho there were others which, judging from the ophthalmoscopic appearances observed, might doubtfully have fallen into the same category, it was impossible to be sure of the origin of the trouble in the absence of reliable histories. The result of such an experience was that the writer's attention was at first directed only to the records of those cases in which the patient had taken 2 drachms or less of quinin in 24 hours. Even then, quite a number of cases were found. The state of affairs which exists; or, perhaps one should say, existed, in certain backward parts of America, where doctors were scarce and the patient helped himself liberally out of a stock quinin bottle, hardly seemed to interest the surgeon who lives in parts where civilization has had time to progress. The opinions expressed by malaria experts of the first rank at the recent meeting of the Society of Tropical Medicine in London, have, however, compelled a modification of the standard thus set up. If military medical officers are going day after day to give from 60 to 90 grains a day, and then to carry on with lesser doses for a considerable time, we must include the cases which have shown toxic amblyopia after the administration of an ounce or more of the drug. Otherwise we shall not have a complete mental picture of the risks incurred. Of one thing there can be

little doubt, namely, that the cases of quinin poisoning reported are but a fraction of those which have actually occurred. The writer has no wish to be an alarmist, but he cannot but feel that the new movement is fraught with perils which it is the duty of the ophthalmologist to point out.

DOSES OF QUININ WHICH HAVE PROVED TOXIC.

We shall start with the cases in which the dose is described as huge, immense, heroic, very large and so on. These are the cases of Behse, Browne (male, aged 34), Bruns (a male aged 16), de Schweinitz (a male aged 40), Fox (a male aged 13), Harbridge (5 sailors, each of whom took a large dose of quinin), Hobby (a female, aged 21), Knapp (two male children, aged 3), Nettleship (a male, aged 29), O'Brien (a male, aged 33), and Williams (a male, aged 14). Everyone of these appears to have been permanently damaged in sight.

Next comes the group in which the dose was about an ounce and upward: Calhoun (a female, aged 10, took one and a half ounces in three days), Guersent (an adult female took 631 grains in a few days), Jodko (1,694 grains of hydrochlorid of quinin in 7 days), Michel (a male, aged 38, took 670 grains in 6 days), Reina (a male, aged 30, took 462 grains at one dose), Roosa (500 grains of cinchona in tincture), Shahan (a female, aged 28, took one ounce of quinin in six hours, during labor), von Kubli (a female, aged 23, took 454 grains), Williams (a male, aged 42, took one ounce in a week). In each recorded case the damage to vision was permanent.

We next take the doses from about 3 drachms to half an ounce: Giacomini (a male, aged 45, took 180 grains), Kalebiakin (154 grains of quinin), Kirkpatrick (a female, aged 30, took half an ounce of quinin), Manolescu (a female, aged 30), took 200 grains of quinin), Parker (a male, aged 43, took 240 grains of quinin), Roosa and Ely (a female, aged 30, took 280 grains of quinin in 6 days), Tyson (a male, aged

23, took 240 grains of quinin in 3 days), Underhill (a female, aged 20, took 200 grains of quinin in pills, but vomited and passed a quantity), Weeks (a female, aged 51, was given 150 grains of quinine per rectum by mistake), and Zani (an adult, took 300 grains of quinin). In all these cases the damage seems to have been permanent.

In the next group we place the doses of about 100 grains: Ayres (a female, aged 7, was given 104 grains in 3 days), Harbridge (an adult male took from 60 to 120 grains in whisky), Kalebiakin (dose 100 grains), Keiper (a male, aged 75, took 120 grains after a drinking bout), Myer (a female, aged 20, took 120 grains in lime juice and water), and Terrien and Aubineau (a dose of 115 grains).

Our next group includes doses of from 40 to 80 grains of quinin: Beydler (a dose of 40 grains of quinin sulphat), Cargill (a female, aged 28, took 60 grains of quinin sulphat), Claiborne (a dose of 60 grains), Fortunati (a male, aged 59, took 46 grains of the hydrochlorid in two doses, and died of fever), Gruening (a female, aged 35, took 80 grains of quinin sulphato in 30 hours), Kalebiakin (dose 80 grains), Kaz (an adult female took 40 grains of the hydrochlorid), M'Gillivray (a male, aged 54, took a 40-grain dose, probably with alcohol), von Speyer (a female, aged 41, took about 60 grains of quinin, of which she vomited half), Weeks (a female, aged 6, was given 80 grains in 3 days), and Welton (a male, aged 31, took 55 grains of quinin in a quart of whisky). Again, as far as can be judged from the records, permanent damage was left behind in each case of these last two groups.

The next group includes the really small doses whose action points to a strong personal idiosyncrasy on the patient's part, except in the case of the child: Bruns (a female child, aged 3, sustained permanent visual damage from a dose of 30 grains of quinin sulphat, given by the rectum in the course of 15 hours), Calhoun (a male adult had temporary amblyopia whenever he took 10 grains of quinin), de Schweinitz (temporary amblyopia followed the

administration of 15 grains of quinin in divided doses in 24 hours), Miller (a sailor, aged 55, took 5 grains daily for 3 or 4 weeks and had temporary amblyopia), Nettleship (a male, aged 26, took 22 grains of quinin in 3 days and was still amblyopic 3 weeks later), Schwabe (a female, aged 33, took 19.29 grains of quinin hydrochlorid for toothache, and her sight was permanently injured thereby), Wood (an adult female suffered from amblyopia after taking 12 grains of quinin). The writer has under his observation a man of 60 who suffers from amblyopia with contracted fields if he takes 2 grain of quinin.

Our last group is full of interest. It contains the cases collected by Conner, all of which either proved fatal, or at least gave rise to very grave anxiety. In Conner's own case, extreme cardiac depression with collapse was met with in a male, aged 27, following the ingestion of 6 grains of quinin. Death seemed imminent, but the patient recovered. It was known that he had always had a strong susceptibility to quinin, an idiosyncrasy which he shared with a brother and a sister. The following cases are mentioned in Conner's communication: Wood's patient showed very alarming symptoms after taking 2 grains of the drug; so did a girl of 13, under the care of Micciche, who had taken 8 grains of quinin. Upshur's case gave great cause for anxiety after a dose of 30 grains, and on a later occasion after a dose of 8 grains Huseman published 4 fatal cases, following the ingestion of 3 drachms, 5 to 10 drachms, 45 grains (in a child of three and a half), and 16 to 20 grains (in a child of two). To this list we must add the cases of the three Chinese coolies, mentioned by Gimlette, each of whom received about 90 grains of sulphat of quinin, in a single dose, on an empty stomach. Two died and the third was very ill with deafness, diplopia and delirium, but recovered.

It must not be forgotten that Berandi noted headache, tinnitus and obscuration of vision as signs met with in his experiments on healthy individu-

als, to whom he administered doses of from 15 to 20 grains of quinin.

ALCOHOLIC AND ACID SOLVENTS.

Attention has more than once been directed to the danger that may accrue from giving quinin either in, or in connection with, an alcoholic or acid solvent. The cases of Harbridge, Welton, M'Gillivray, Keiper and Myer are all to the point. Their interest lies in the great severity of the symptoms at the time of the first poisoning, and in the extent of the permanent damage done. When one considers that these were all adult males, and that the doses taken were none of them really large (respectively 60 to 120 grains, 55 grains, 40 grains, 120 grains and 120 grains), one cannot easily resist the conviction that great care should be taken to regulate the doses of this powerful drug, when giving it in combination with solvents. The plain indication would appear to be that we should not let the quantities administered get out of hand, until we have made sure that the patient has no idiosyncrasy for quinin. It may be said that the same remark could be made with equal justice about quinin being given in any other form. Whilst this is quite true, the danger seems to be accentuated in dealing with dissolved and concentrated quinin, and therefore extra caution is demanded under such circumstances.

There are points of interest in connection with the *repetition even of small doses* of quinin. Thus, Miller's patient, a ship's captain, aged 55, took only 5 grains a day with the exception of one occasion on which he doubled the dose. Yet, after 3 to 4 weeks of this treatment, he had developed typical signs and symptoms of quinin amblyopia. It is true that he was a heavy smoker, but the ophthalmoscopic appearances presented, the absence of central color disturbances, the restriction of his visual fields, and the recovery of central vision, when quinin was stopped, show clearly that it was a case of poisoning by this drug.

It is of course possible that tobacco contributed to the toxic effect, just as we may see optic atrophy progress in a glaucomatous eye after the complete re-

lief of tension, owing to the existence of some other hitherto unrecognized cause of autointoxication. That the latter is the correct explanation in such cases would seem to be clear from the fact that the removal of the accessory cause of autointoxication will sometimes arrest the atrophic process started by the glaucoma. It is possible that the action of quinin may be accentuated in the same way by that of one of the other drugs, which produce toxic amblyopia.

An extraordinary and somewhat unexpected light would appear to be thrown on this subject by Schwabe's case, of a woman of 33, who had suffered from quinin amaurosis for 19 hours, and from amblyopia for three years after a dose of 19.29 grains of muriat of quinin, administered for toothache. Whenever she took a cup of strong coffee or strong tea, the retinal vessels could be observed to become powerfully constricted, and the fields of vision narrowed down almost to the fixation point. The maximum effect was attained in one hour, and the trouble had passed away in two. There was no evidence of permanent loss of central visual acuity, of central color vision, or of the area of the fields of vision, despite the fact that the patient daily indulged in these beverages, thus frequently repeating in her daily life what one would have thought was a hazardous experiment.

Caffein or thein is known to be a cerebral stimulant, and as such has been advocated in the treatment of quinin amblyopia, in spite of the fact that more than one writer has suggested, possibly on not very good evidence, that it may itself be a cause of toxic amblyopia. Schwabe's careful observations are calculated to revive such suggestions, and to indicate a measure of care in our use of the drug in quinin amblyopia. It is possible that the cases in which it acts disadvantageously are instances of individual idiosyncrasy, but it would be unwise to forget the lesson they teach. The writer, however, knows of one case in which the administration of strong coffee definitely resulted in the amelioration of the signs and symptoms of quinin amblyopia in a patient who is so sensitive to

the drug that he presents contracted fields and interference with vision after so small a dose as two grains of quinin sulphat.

De Schweinitz lays stress on the *liability to relapse* shown, after the exhibition of small doses of quinin, by patients who have already suffered from poisoning by large doses of the drug; and draws attention to the fact that Knapp and Nettleship had both made similar observations. He himself has noted an exactly similar sequence of events in animals. Manolescu, in his very careful treatise on quinin poisoning, has drawn special attention to this phenomenon, and has urged that in the case of all patients, who have been known to have had quinin amblyopia in the past, the greatest care should be exercised, not only with regard to the use of the cinchona alkaloids, but also to that of any of the other poisons, such as alcohol and nicotine, which are known to be liable to produce amblyopia in any form.

The literature available contains at least two highly illustrative cases, which demand a short notice. Weeks' patient, a girl of 6, was given 60 grains of quinin one day by the rectum; and 20 grains more in the next two days. She suffered from severe amaurosis, but eventually recovered her central vision, tho the fields remained much contracted. Twenty months later she was given a single dose of 10 grains of quinin in one day, with the result that the signs and symptoms of poisoning of the optic nerve and retina returned with great intensity. Altho she again recovered to some extent, a good deal of the damage done on the second occasion was permanent. In Hobby's interesting case "very large doses of quinin had been required to produce cinchonism" in the first instance; but when, on a later occasion, 20 grains of the drug were administered, the patient had a very severe return of amblyopia; which affected both her eyes, whereas in the original attack, the right had nearly escaped.

METHOD OF ONSET AND COURSE OF ATTACK.

A very careful analysis has been made of the reported cases of quinin poison-

ing, with a view to ascertain the methods of appearance and of development of the signs and symptoms of this condition. This survey has revealed very great differences in different cases. This is only what might have been anticipated, in view of the facts that the size of the dose and the method of taking it varied very widely, as did also the age, weight and sex of the patients, and the measure of individual idiosyncrasy. Even this list does not exhaust the disturbing factors present. Many of the cases are very indifferently noted, and important points are slurred over or are altogether omitted.

It is quite obvious that in not a few cases the observers failed for comparatively long periods to grasp the true nature of the patients' troubles, and continued to pile up the doses of quinin when the drug should have been at once and completely withdrawn. Some of the most melancholy of such cases are those in children, in whom it seems to have taken days before it was recognized that the little patients were blind. It is clear that in many of the cases, the onset of acute cinchonism was mistaken for a recurrence or a development of the disease for which the drug was given. If there is one thing that should be written in heavy type across the literature of this subject, it is that *quinin poisoning is a condition which can be diagnosed with ease and certainty by the least experienced practitioner, and that mistakes, such as those which abound in these records can and should be erased from medical experience.*

The administration of medicinal doses (10 to 15 grains) of a salt of quinin gives rise in normal subjects to a train of symptoms, which is so constantly met with and of such slight clinical significance, that it is best described under the term "cinchonism"; reserving that of "quinin poisoning" for the more serious form of the same condition, which attends the ingestion of large doses, or that of comparatively small doses in very susceptible subjects.

CINCHONISM.—Soon after taking the quinin, the patient complains of ringing in the ears, deafness and a feeling of fullness in the head. Headache, which

may sometimes be very severe, may be experienced. Giddiness may supervene, and the patient may even stagger in his walk. Often he will wish to lie down, and feeling drowsy, he may quickly fall asleep. All these symptoms are transitory and as a rule have passed away completely the next day; always provided, of course, that further doses of the drug are withheld.

QUININ POISONING.—In a very large number of the recorded cases in which a single large dose was given, the signs of this condition are described as coming on "the next day"; or "after the patient awoke" from a heavy sleep, or from a comatose condition which followed the dose. There can be no reasonable doubt that the real evidence of the quinin intoxication was forthcoming much earlier, had it been looked for, but was missed owing to the patient's drowsy condition, or because it was not anticipated. Often, the striking symptom that first arrests attention is partial or complete blindness. The accompanying deafness is usually made less of, because as a sign of cinchonism it is so familiar as to have bred contempt. When the drug has been more gradually administered, failure of sight may again be the first sentinel symptom. As will be seen later, the interference with vision may be noticed within the first half-hour or even quarter-hour, and may steadily and rapidly deepen.

DIMINUTION OF CENTRAL VISUAL ACUITY.—The onset of central blindness is so striking a symptom that it compels the attention of the patient in a way that no other does. If one were to judge from the text of the reports alone, one might reach the conclusion that the onset of blindness was often sudden. If a surgeon were on the lookout for the well known symptoms from the first, it seems more than probable that he would find the loss of sight heralded by amblyopia and contraction of the field of vision. This has certainly been the case in a few instances, where close observation was possible.

Much depends on the dose and method of administration of the quinin. When a single large quantity of the drug has been

taken, interference with vision has been noted in a few cases in from a quarter to half an hour (de Schweinitz, Kalebiakin's three cases, Manolescu, Myer); a number of other observers are content to describe the blindness as coming on "soon after" the dose was taken (Harbridge, Nettleship, Roosa, Trousseau and Pidoux, Underhill and von Speyer). In one of Weeks' cases, in which the dose was very large, the period mentioned was three hours; in Schwabe's patient five hours elapsed, but the dose was here under 20 grains.

We come last to the patients, who first noticed blindness on awaking next morning, or on recovering consciousness after several days of coma; with them we have already dealt; it is impossible to say when their symptoms commenced. In considering the patients in whom the administration of the medicine was spread over a number of days, it is much more difficult to obtain anything like scientific accuracy, but it is quite clear that in them the drug produced a cumulative effect.

In saying this, it is not implied that the concentration of quinin in the blood increased steadily, for such a suggestion would be contrary to the known facts, but rather that the continued toxic action sufficed in time to bring about a condition of blindness which comparatively large earlier doses failed to do. It is of interest to note that in two cases, the final extinction of vision was instantaneous; so much so as to suggest sudden vasomotor constriction as its cause. Thus, Browne's case said his "sight went out as if you had turned out the gas," while in Michel's patient it is recorded that "vision suddenly went out."

The *development of the blindness*, from the moment of its first appearance until it became complete, varied enormously. A few never attained to complete amaurosis but were seriously alarmed by the degree of their amblyopia. Cargill's patient could not find her way about her room; whilst Welton's patient stumbled over objects on the floor and complained of hazy vision. In a number of the worst instances, complete amaurosis appears to have been very rapidly established. There can be but little

doubt that a large number of cases, in which a moderate amblyopia is present, fail altogether to be recorded or even to attract attention.

Then again, the *duration of the blindness* varied enormously. In a few, it had begun to pass off in from 14 to 24 hours (de Schweinitz, 14 hours; Schwabe, 19 hours; Keiper, 22 hours; Harbridge, 24 hours). In a number of others the period varied from 2 days up to 2 weeks (Underhill, 48 hours; Collins, Kaz and M'Gillivray, each 3 days; Weeks, 4 days; Manolescu and Weeks, each 5 days; Ayres and Tyson, each 7 days; Parker and Williams, each 8 days; Bruns, 13 days, and Behse, 2 weeks). A few still longer cases are on record, in some of which the exact date is not specified (Shahan, 18 days; Michel, 32 days; Reina, 8 weeks; Voorhies, 10 weeks, whilst in Calhoun's case the patient was lost sight of still totally blind 10 weeks after the dose). In some of the animals he experimented on, de Schweinitz found the loss of vision still total at the expiration of two months.

It is one thing to estimate, as we have just done, the period at which vision commenced to return; it is quite another to determine when full sight was restored. A careful survey of the records cannot fail to suggest the gravest doubts as to whether complete restoration of vision ever occurs in an amaurotic case. The question is largely one of degree, of the care with which the examination is made, and of the standard accepted as "perfect vision." This will be taken up more fully under the headings of light sense, and of the fields of vision. Meanwhile we may mention that in one of Kalebiakin's cases, the complete restoration of vision was said to have been "almost immediate." Manolescu and Zani each reported full recovery in 14 days; Nettleship in 3 weeks; Kalebiakin in 1 month; Underhill and Williams each in 6 weeks; Parker in 3 months; Brown in 4 months; Weeks in 1 year, and Tyson in 14 months.

In addition, there are a number of records of even longer periods, but it is probable that these represent the times at which the cases were again seen,

rather than those at which recovery could have been claimed to have occurred. A very interesting feature of some of the cases is that vision improved up to a certain point, and then went back again. A late second improvement may again be noted (Demicheri, Goldzieher, Kirkpatrick, Mellinger, Michel, Roosa, Ely and Weeks). Finally, it remains to mention that the records clearly show that in a large number of the cases a visual acuity of 6/6 was never reattained, whilst the silence on this subject of many of the reporters is still more significant. Even if central vision be alone accepted as the basis of our judgment, which it obviously should not be, the writer is at wide variance with the optimistic views of Manolescu as to the ultimate restoration of vision after quinin amaurosis. The reasons for this opinion will be better appreciated after a perusal of the next few sections.

ALTERATIONS IN LIGHT SENSE.—It seems probable that a defective light sense would have been found in quite a large number of the patients, who have suffered from quinin amaurosis, if only the examination had been sufficiently careful and exhaustive. It is significant that our sources of information on this head come from the observations of comparatively few writers, most of whom were ophthalmologists of exceptional note. It is quite obvious that in the majority of the cases, no careful estimation of the light sense was made; indeed, the possibility of a defect in it does not often seem to have been taken into account. In any case it would be difficult to estimate such a defect in the early amblyopic stages, and nearly all the notes refer to a late period of the affection. An exception to this rule is found in Nettleship's patient, who "found he could stare at the sun without inconvenience" shortly after the commencement of return of vision. Two months later, he complained of seeing badly in a bright light, and of a mist before his eyes in the early morning. Weeks observed a marked diminution in light sense in both of his cases of quinin poisoning. One of his patients complained bitterly of reduction in this sense up to the day of her death; whilst in the other light percep-

tion was diminished and light adaptation slow 35 years after the origin of the poisoning. Roosa and Ely's patient was reported 3 years after the intoxication, as never having seen well since she took the quinin. She "felt as if a veil were over her eyes." De Schweinitz' patient was markedly night blind after 8 months, and his light sense was reduced to 0.6 of the normal. Welton's case resembled this in being badly nyctalopic after 2 years; although it was doubtful whether he had ever been absolutely amaurotic. Other instances of damaged light sense are recorded by Ayres, Bruns, Manolescu, Panas, Parker, Schwabe, Shahan, Williams and Zanotti.

ALTERATIONS IN THE VISUAL FIELDS.—A constriction of the visual fields is probably the most constant sign of quinin poisoning of the eyes. As the amaurosis or amblyopia passes away, it is found that the patient is considerably hampered by the loss of his peripheral vision. He stumbles over objects on the floor, or has difficulty in finding his way about a room. This defect continues when the central vision, as measured by the test types, has returned to normal, and may give rise to very grave inconvenience. On the other hand, when the dose has been comparatively small, rapid improvement may take place, and after a period of months or even of weeks, the recovery may be so great as to lead the patient and his medical adviser to believe it perfect. Without denying that such a happy result may sometimes be attained, it is quite certain that such cases constitute the exceptions and not the rule. In a very large number of instances, a permanent and often progressive reduction of the visual fields has been recorded. This is sometimes so extreme as to constitute "tube vision," (de Schweinitz, Kalebiakin, Schwabe, Tyson, von Kubli, von Speyer, and Weeks), whilst in quite a large number of cases the defect, though less extreme, is yet well marked.

Improvement may go on taking place for months and apparently even for years (Tyson and Weeks). On the other hand, the fields may enlarge up to a certain point and then become nar-

rowed again. It is possible that these secondary deteriorations are due to the unwise administration of quinin on a later occasion, as happened in Weeks' case; or to the abuse of some other toxic drug, such as caffeine (Schwabe).

Calhoun has recorded the case of a prominent physician, who found that 10 grains of quinin made him amblyopic, whilst repetitions of the dose caused alarming symptoms. The writer has under his care a man of 60, in whom a dose of 3 grains of quinin will at any time cause marked constriction of the field. It is probable that such cases as those above quoted explain the late deteriorations of the fields which are on record, but it is of course quite possible that the latter may occur independently of the administration of toxic drugs.

It has from time to time been suggested that the typical visual defect in quinin poisoning leaves a horizontally elliptical field, the upper and lower areas being more encroached upon than the lateral ones, (Harbridge, Knapp, M'Gillivray and Welton). Again, it has been suggested that one area of the field, as the temporal or the nasal, tends to clear up sooner than the others. A survey of all the records lends little support to such suggestions, the contraction being apparently usually concentric. Some very anomalous fields have been noted; thus Mosso described a paracentral scotoma, which was first absolute, later relative and finally annular; whilst Bietti, Galezowski and Jodko all record instances of central scotomata.

ALTERATIONS IN THE PERCEPTION OF COLORS AND IN COLOR FIELDS.—In those cases in which the central color vision was tested at a very early stage, it would appear to have been uniformly deficient or absent. Improvement, however, set in fairly early in most cases. In some, the central color vision again became perfect (Browne, Bruns, Cargill, de Schweinitz, Schwabe and Williams). In others, the recovery was less complete, (Keiper, Tyson and Williams). Shahan's case was totally blue-green blind after 3 months. One of Weeks' cases was green blind after 2 years, but a second small dose of quinin had been given in the interval. Knapp's patient was at

first totally color blind. This gave place to red-green blindness, then green and grey were confused, and finally, at the end of five years, complete color perception was reestablished. The extraordinary variability in the course of the recoveries is illustrated by comparing the last case with that of Cargill, whose patient could not distinguish colors on the ninth day, but could do so on the forty-fifth. Still more striking is Schwabe's case, in which there was no color vision 43 hours after the dose, while on the 5th day, the central color perception was good. The notes on the state of the color fields are scanty, but they were reported to be greatly contracted by Browne, de Schweinitz, Stasinski and Welton.

DILATATION OF THE PUPIL.—In the great majority of the cases of quinin poisoning the pupil is described as being fixed and dilated, the latter often to an extreme degree. No observations appear to have been made as to the exact time when the pupil begins to enlarge, but from his observation of a very susceptible case, the writer thinks that this probably takes place at a very early stage. It is a phenomenon of the greatest importance, inasmuch as it enables even one who is not an expert to get a good and easy view of the fundus. The combination of a dilated pupil with the typical ophthalmoscopic appearances should leave no doubt as to the rôle of the drug in the causation of blindness. As the case progresses, and the patient recovers vision, the extreme dilatation of the pupil passes away, and the normal movements are reestablished.

But it is probable that some enlargement remains permanently in quite a number of the cases, nor is it certain that the movements wholly regain their former activity and rapidity. In Cargill's case the pupils reacted slightly to light and accommodation on the thirty-eighth day, and well on the seventy-eighth. The pupils were slightly dilated and the iris reaction was sluggish in Reina's patient, 6 months after the poisoning. The same held true for the right eye in Kirkpatrick's very interesting case, nearly a year after the dose was taken, whilst the left eye showed a widely dilated and fixed pupil. A similar state of affairs has been

recorded after still longer periods, viz., 1½ years (Harbridge), 2 years (Weldon), and 5 years (Knapp). In Bruns' two cases there was slight dilatation of the pupil 10 and 17 years respectively after the poisoning.

A few points of interest remain to be recorded. In Knapp's case above mentioned the pupils responded to accommodation but not to light. In Hobby's patient, the pupils though widely dilated, reacted to eserine. Contraction instead of dilatation of the pupil has been described by Roberts, whilst Shahan noted active elliptical pupils on the 18th day after a very large dose of quinine had been taken.

THE OPHTHALMOSCOPIC CHANGES OBSERVED.—The most characteristic of these are pallor of the optic discs, and extreme contraction of the arteries and veins of the retina. These two phenomena recur with monotonous regularity in all the reports. The appearance of the nerve head is such as to suggest advanced atrophy; the pallor is striking, and the edges of the disc stand out by contrast with great sharpness against the surrounding fundus. The retinal blood supply is so greatly diminished that its vessels, and especially its arteries can sometimes be traced but a very short distance out from the disc edges. The next most frequent of the signs observed have been those of a cherry spot at the macula and of retinal edema (Berger, Buller, Gruening, Reina, Seligsohn, von Speyr, Weeks and Zani). Reina and Weeks differ from the other observers in describing the macula as reddish, and as brownish-red respectively. Weeks explains this peculiarity in color as due to the absence of edema at the fovea. He points out that the absence of the vivid cherry color, found in obstruction of the central artery, is due to an ischemia of the choroid as well as of the retina, the former resulting from the action of quinine on the choroidal vessels. The absence of a free flow of arterial blood, in his opinion, causes the fovea to lose much of the vividness of color it would otherwise possess. Nettleship, in a case seen two months after the poisoning, found a certain degree of retinal edema, whilst Gruening observed the characteristic macular phenomenon and the surround-

ing edema disappear in 9 days. A few surgeons have recorded a thickening of the arterial walls presenting itself in the form of white streaks which run along the vessels (Bruner, de Schweinitz, Moulton, Parker, Reina, Seligsohn, Terrien and Aubineau, Uhthoff and von Speyer).

Fundus changes, indicative of inflammatory action, have been recorded by a few observers. These include retinal changes (Buller, Demichieri, Keiper, Parker and Zanotti); hazy edges to the papillae (Ballantyne, Bietti and Roberts); and choked disc (Dickinson, Zani and, doubtfully, Terrien and Aubineau). The columns of blood in the retinal vessels were observed to be broken in Parker's case; and retinal arterial pulsation was recorded by Buller and Manolescu; Parker reported a thrombus in a branch of a retinal vein in each eye; Kirkpatrick found one nasal artery in each eye much thickened, almost obliterated and partly covered by a hyaline mass as it crossed the edge of the disc. It is possible, if not probable, that some of these rarer changes are accidental, and have no connection with the quinine poisoning.

There is a point of very great interest, which we shall merely mention here, but hope to return to at greater length in discussing the etiology of the affection, viz., that there are indisputable cases on record, in which quinine amaurosis is present in the absence of the characteristic ophthalmoscopic appearances which we associate with this condition (Ballantyne, Webster Fox, Garofolo, Hamlich, Jodko, Kaz, Mantendam, von Graefe and Wilbrand). This observation increases in interest from the fact that a similar experience has been met with in dogs (de Schweinitz).

UNUSUAL EYE SYMPTOMS.—The following unusual phenomena have been met with:

(1) *Unilaterality of the affection* (Browne, Hobby, Kirkpatrick, von Graefe and Westhoff). These cases all differed from one another, but at the same time had this in common that some one or more of the features of the condition (dilatation of the pupil, ophthalmoscopic appearances, central visual acuity, visual

fields, etc.), were better marked in one eye than in the other.

(2) *Alterations in Sensation.*—An anesthesia of the conjunctiva was met with by Barabaszew and Belsky, and of the conjunctiva and cornea by Voorhies, whilst Parker's case presented a hazy and hyperesthetic cornea. Both Barabaszew and Parker noted hypotonus associated with the above conditions. In Stasinski's case also a lowering of the ocular tension was present. Tiffany records a case with hypotonus.

(3) *Affections of the Muscles.*—Atkinson observed lid spasm; Browne, Claiborne, Diez, Gimlette, Knapp, Peña and Wilbrand record evidence of pareses or paralysis of the ocular muscles; Mosso met with paresis of accommodation. Nystagmus was observed by Jodko, Knapp, Roosa and Williams; in Knapp's case it was vertical and vibratory and was associated with periodic divergence of the right eye. De Schweinitz also observed nystagmus in animals poisoned by quinin.

AURAL SYMPTOMS.—It has been mentioned that deafness is an early and troublesome sign of quinin poisoning. As a rule, it passes off quickly and completely. Williams, however, records a case in which the hearing was never wholly recovered, and it seems likely that many more such have been missed. In view of what has been found in connection with vision, and bearing in mind the similarity of the attack by quinin on these two special senses, one cannot help thinking that, if the hearing of a number of these cases were very carefully tested, evidence would be forthcoming of some permanent damage in a definite percentage of them. With the deafness is associated a variable and often distressing amount of tinnitus, and a feeling of fullness of the head. More rarely there is definite headache, which occasionally may be extremely severe. Giddiness, or dizziness, makes a not very infrequent appearance in the notes, tho probably it is a symptom, which is often missed, or belittled. It remains to mention Welton's very unusual case in which there was no tinnitus or deafness, and in which the patient's mental condition remained clear,

although the vision was profoundly affected. Roosa and Ely's experience was allied to Welton's, inasmuch as aural symptoms were conspicuous by their absence, but on the other hand, delirium was present.

GENERAL SYMPTOMS OF QUININ POISONING.—In a number of the cases of quinin poisoning by massive doses, one of the earliest notes concerns the recovery of the patient from a state of coma or of profound sleep, and the observation, at this time, of blindness, or at least of serious visual defects. This suggests, either that the patients fall off to sleep as a result of the administration of the drug, or that the condition is often so serious as to render routine clinical observation difficult. In any case, it is clear that very heavy sleep, and often a condition of coma, which may last for several days, are to be expected. More rarely, the patient is described as answering slowly, or as being in a state of drowsiness or of stupor (Manolescu). On the other hand, an irritable cerebral condition may be present characterized by troublesome delirium or by visual hallucinations (de Schweinitz, Gimlette, Laveran, Parker, Roosa and Ely). Manolescu records a case in which there was want of control over movements, and Myer one in which the patient lost the use of his arms and legs. Gruening's patient had a convulsive attack, whilst comatose. Respiration may be very shallow and death may be brought about by respiratory embarrassment. Cardiac failure is another grave danger, which may lead to fatal results. Gastro-intestinal symptoms are not wanting; vomiting and diarrhea are frequently mentioned. The former may come on at an early stage, and be obviously due to direct irritation of the gastric mucous membrane; later it may be very troublesome and is then probably cerebral. The stools and urine may be voided unconsciously.

It will be clear from the foregoing statements that it is only too easy to confuse the signs and symptoms of quinin poisoning with those of the various severe fevers, for the cure of which the drug is administered. From a perusal of the available records, it is obvious that this has been sometimes done, with the

result that the drug has been pressed at a time when it should have been altogether withdrawn. The tragedy of such a mistake is all the greater in children, in some of whom it would appear that it took days before it was discovered that the so-called treatment was the real cause of the trouble. These little patients paid by lifelong blindness—the

penalty of the doctor's error. Such disasters should be relegated to the limbo of an unforgotten past. Any hesitation as to the diagnosis should be banished from the mind by an examination of the widely dilated pupils and by an observation of the extreme retinal ischemia, which is almost uniformly present.

(To Be Continued)

NEUROFIBROMATOSIS OF THE ORBIT.

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History of a case of multiple neurofibromatosis, with illustration showing orbital involvement. Operation and result.

Neurofibromatosis of the orbit is rare, especially with simultaneous involvement of the optic nerve. In the following case of general neurofibromatosis with orbital involvement, two masses were removed from the supraorbital nerve and one mass remains deep in the orbit, which may or may not arise from the optic nerve.

The following clinical picture points to optic nerve involvement: (1) Marked exophthalmos and ptosis. (2) Mobility of the eye is good in all directions. (3) Vitreous opacities. (4) After tumor masses were removed from the supraorbital nerve the pain disappeared. To strengthen our suspicion that the tumor is not attached to the optic nerve the following findings are of importance: (1) Vision 4/10 increased to 4/4 with correction. (2) Absence of optic neuritis and atrophy. (3) Absence of high grade or even moderate venous engorgement. In a case so dubious, a positive diagnosis can only be made when the symptoms become more definite, or enucleation with section reveals the true origin of nerve attachment, and whether it is intradural or extradural to that nerve. Personally I am strongly inclined to the extradural non-optic nerve diagnosis.

CASE.

Mr. P. U. Age 42 years. Married. Occupation, janitor. Habitat: Thru northern Michigan. Referred by Dr. Jones of Bay City. Diagnosis: General neurofibromatosis (Recklinghausen's disease, neuroma plexiforme). Diagnosis based on the pathologic report and the clinical findings.

Previous health has always been good excepting the diseases of childhood. No history of head injuries. Denies all venereal diseases. Weight 150 pounds; height 5 feet 11 inches. Has lost some weight during the past couple of years. Urine negative. Leucocytes 7800. Erythrocytes 4,000,000. Hemoglobin 75% (Sahli). Wassermann negative. Physical examination negative for chest and abdomen, except scars resulting from previous operations. No paralysis but there are restricted movements due to scar tissue contractions, the results of the operations. Some tingling in the fingers of the left hand. The left forearm has had many growths removed four or five times.

Character of the Patient.—Clear minded, intelligent, not neurasthenic.

Chief Complaint.—Intense pain in left eye, more or less continuous, with quies-

cent intervals of partial and complete relief, to be followed by an onset of pain increasing in intensity till it crushed his morale and produced a state of abandonment where he gladly would part with the eye to obtain relief. But all attacks did not rise to this grade in severity. There was no discoverable cause for the paroxysms. On his own initiative he suggested enucleation if necessary.

HISTORY OF PRESENT CONDITION.—Began about 23 years ago, with removal of superficial tumors from the forehead and left wrist. Since then he has tried many forms of non-operative treatment without success. Six years after the first operation he again had some masses removed. Now he has the tumors removed every two years but they are becoming more frequent, larger and more deeply situated. For the past four years has had increasing pain in the left eye and toothache. But during the last six months the toothache has disappeared, altho pain in the left eye has become excruciating.

The exophthalmos and ptosis began very gradually two years ago. At present, 3 mm. of conjunctiva is exposed when the lids are closed. The photograph shows the marked exophthalmos and proptosis with the margin of the lower lid of the right eye on the same level with the margin of the upper lid of the left. Palpation readily causes tenderness of the tumors. These masses are felt beneath the upper lid between the ball and the margin of the supra-orbital arch, in the midline; and seem to be 10 mm. by 10 mm. in size. No increase of tension. Vision: R. 4/5 and L. 4/10, increased to 4/4 by —.50 Sph. with —.50 Cyl. axis 105°. The fundus of the L. showed the disc slightly pale and veins little enlarged. These were the only findings.

OPERATION.—Ether anesthesia. Incision thru shaved eyebrow. Dissection down to first tumor which was excised. Second mass, slightly deeper, was also removed. Both of these were attached to the supraorbital nerve. The third mass was deeply situated. It could be touched with the finger, but no attempt was made to remove it, because of its

depth and possible attachments and the dangers of dissecting, by the sense of touch. The wound was sutured with horse hair.

Recovery uneventful. No pain or tenderness.

RESULTS OF OPERATION.—(1)—Absolutely no pain. (2) Some reduction of



FIG. 1.

Neurofibromatosis of Orbit. Campbell's case showing eyeball pushed down and forward by growth in orbit.

the exophthalmos and proptosis. (3) Two neurofibromata removed. (4) The knowledge that removal of the remaining tumor will necessitate enucleation. This mass remaining in the orbit apparently causes no symptoms, except the exophthalmos and proptosis.

Subsequent result of operation, ten months later.—The pain has not returned to the left eye, but intense itching at the scar site occurs in paroxysms. Headache when using eyes for close work over a prolonged period, but this stops when the eyes have been given a rest. The exophthalmos and proptosis have somewhat increased. No contraction of

the visual field. Patient sees several spots before the left eye. The fundus shows nothing; but the vitreous has several small rapidly precipitating, dark floaters. Vision 4/6 correction with a plus 0.50 sph. and a plus 0.50 cyl. axis 30° gives 4/4. Tension not increased.

The corneoscleral margin is exposed about 5 mm.; this is an increase of 2 mm. over what was exposed before the operation.

The prognosis as I see it, is ultimate destruction of the eye by a keratitis due to corneal exposure.

CONJUNCTIVITIS OF THE FORNIX.

BY DR. JUAN SANTOS FERNANDEZ.

HAVANA, CUBA.

This paper calls attention to a condition characterized by serous infiltration and protrusion of the conjunctiva of the fornix; and reports an illustrative case.

The fornix, as is well known, forms that portion of the conjunctiva (from *conjuguere*, to unite), lying between the bulbar and the tarsal conjunctiva, the *culdesac*, of the French authors and the *fornix* of the anatomists.

Surely the division of the conjunctiva into four regions, tarsal, bulbar, fornix and lacrimal, responds to histologic differentiations, as well as anatomic, functional and pathologic ones, but the anatomic interest is almost nil. Some authors study separately these four regions and on describing the fornix call attention to the dangers that it may present, as in *ophthalmia neonatorum*, in some cases of which the secretion may be accumulated in considerable amount thus preventing the curative effect of the drugs used to combat the disease.

Outside of what we have stated, few authors really call any attention to the fornix as the seat of any disturbance, and as we have been observing in Cuba for a long time a special alteration of not well defined status but with its own characteristics, in the fornix, we try now to describe it; and want to call attention to this pathologic entity that we have named conjunctivitis of the fornix. Not a few times it has been called trachoma and some cases have been operated as such without any injury to the patient, even if the cases were not of trachoma,

because it is an inflammatory disease that can be attacked in different ways.

The conjunctivitis of the fornix is a lesion confined to the region mentioned. We believe that these cases must have some relation, not yet well defined, to the climate of this country, or to the general condition of the child suffering the disease. The anemia thought to be frequent in these patients has not been proved by the blood findings. In regard to this we have published a long time ago some statistics of eye patients in Cuba; and we proved that trachoma was not very frequent here until after the war, and the coming into Cuba of many thousands of immigrants. We have also written more than once concerning the existence of false trachoma¹ and have shown that in spite of the strictness of the health authorities in the ports, especially at Havana, some cases of trachoma enter the Island of Cuba. The diagnosis of trachoma is really very difficult and only by the observance of the course taken by the disease can it sometimes be diagnosed. On that account we are always cautious when giving a diagnosis of trachoma especially when there is some legal matter involved in that connection.

Axenfeld² was undoubtedly right when he said that the diagnosis of some troubles of the conjunctiva was a difficult matter, met only by means of a meth-

odic examination, that should be made for some time before giving out a diagnosis that there is some especial disease or a combination of diseases. In the same way there have been some opinions concerning whether trachoma is due to one cause only, a germ for example, or to several causes. These doubts cease in the case of a special infection, like the one produced by the Klebs-Loeffler bacillus, or the gonococcus.

Referring again to the conjunctivitis of the fornix, although our cases have been seen by the hundreds, we describe only one case from the many we have seen lately, chosen because it is typical.

CASE.—J. N. M., a great grandson of a lady that had been operated by me for cataracts in 1875, was brought to me by his father. The patient was a boy nine years old, and the father told us that for the past two years he had been noticing something abnormal about the boy's eyes. No pain, and very slight lacrimation at times. But what called the attention of the parents was the constant twitching of the eyelids, lasting sometimes fifteen minutes, and done in spite of the efforts made by the boy not to do it. We shall afterwards give our opinion of this twitching. In order to ascertain the cause of it, we everted the upper lids and found the conjunctiva of the tarsus in normal state with slight asthenic decoloration.

On asking the boy to contract the lids (the upper one being everted), the phenomenon that was seen is the pivotal axis of this paper. When the patient made the contraction asked, there was produced an eventration or hernia of the conjunctiva of the fornix, that appeared like a small white tumor in between the space limited by the lower border of the cartilage and the eyeball, and this proved to be the conjunctiva of the fornix, inflamed and edematous, of pale red color and showing some venous predominance or asthenic aspect, the size being that of a small horse bean.

There was a very slight secretion and the bacteriologic examination revealed no special microorganisms, outside of the common ones found in the

normal conjunctiva, like Koch-Weeks' bacillus, streptococcus, the diplobacillus of Morax-Axénfeld and other germs, none of which had any relation to the present disease.

The father of the boy had seen another specialist who had recommended an intervention, believing the disease to be trachoma; and the patient had to leave the Island to go north. We told him that if the trip was to be done immediately, the best course would be the rapid surgical intervention, but that in case the trip was going to be delayed, the medical treatment would be satisfactory; and as the parents decided to delay their trip we began to treat the patient locally. The belief that the patient was anemic, and the fact that he had had an operation on the nose some time before, made us order the blood examined several times. The average results were: Red corpuscles, 5,200,000; hemoglobin, 85%. The fact that the same results were obtained in many cases allowed us to discard anemia as a probable factor in the etiology. The case described was cured very rapidly with the medical procedures used.

The two salient symptoms of the "Conjunctivitis of the Fornix," are the twitching of the eyelids, and the protrusion of the inflamed or infiltrated fornix. The latter, it can be clearly seen, is not a conjunctivitis, but a serous infiltration of the conjunctiva of the fornix, of possible lymphatic origin, giving rise to the swelling noticed³. The small tumor appears in the lax fold formed by the conjunctiva of the posterior culdesac. That is why it appears as a swelling or tumor in the space between the cartilage and the eyeball. As there is no great inflammation, there are no inflammatory symptoms, and the effect produced by the inflamed fornix is like a foreign body; without any pain and exciting only the contraction of the lids, this being the first symptom noticed in the patients, who are generally children.

What the authors say regarding the structure of the culdesac is in favor of our explanation that the matter is an

infiltration of the fornix. It is admitted that the conjunctiva of that region extends from the zone where it is adherent to the tarsus, to the level where it comes in connection with the periscleral tissue. The upper or lower border of the cartilage, as we have said, allows us to give a precise and fixed limit to the conjunctiva of the region mentioned. The macroscopic aspect is changed naturally from this point on, and being smooth at first becomes afterwards more rugged and less uniform, there being some folds that have a horizontal direction and that are well marked in some pathologic conditions, the same not being present in fetal life. These folds greatly facilitate the motions of the eyeballs, and they are taken into account when one is going to operate. Through the upper fold the extirpation of the lacrimal gland is done and through the lower one the levator palpebral muscle is sought when the Motais or Parinaud's operation for ptosis is to be practiced; the same way also when the extirpation of the culdesac is needed.

The most important reason we have to pay attention to this subject is to differentiate it from trachoma and as we have already said, since the beginning of the American intervention in Cuba, in 1899, we called attention to the frequent errors made in diagnosis, for many such cases were taken as trachoma¹. We believed that these errors, altho not to be approved scientifically, were beneficial from a sanitary standpoint as in that way no true cases of trachoma could be smuggled in, and because we believed that those children were more amenable to contract trachoma than healthy ones. From a scientific point of view, however, we could not agree with the sanitary opinion, for most of those cases were really

not due to trachoma; and besides, in Cuba there is very rarely seen those severe scrofulous conjunctivitis that are so intense in the cold climates.

In Cuba the lymphatic manifestations of the eyes are very moderate and tend to appear as if due to anemia, as we thought at first, but have changed our view since then. The languor that is believed to be very common here and in all tropical countries, and that has been thought to have influences even of a psychic strain, as stated by a distinguished Argentine writer (Ingenieros), but combated by Dr. Diego Tamayo and others, has no very marked influence. But there must be allowed some influence to the extreme heat, the nervous excitation and the mixture with some inferior races, which to some extent are factors to be taken into consideration.

CONCLUSIONS.—1. The affection of the fornix we have described appears to be more of an infiltration than an inflammation. The infiltration being in the conjunctiva of the culdesac, more loose there than in other places and probably due to a slight manifestation of lymphatism.

2. It is absolutely unrelated to trachoma in its various manifestations.

3. It is not contagious, but might safely be so considered, because even if it does not transmit any infection, the patients are subject to being infected with great ease. This fact has been the cause of many errors of diagnosis.

4. The local treatment is the same as for any simple conjunctivitis, preferring the antiseptics in order to avoid the secondary infections. The general treatment has to be dependent on hygienic rules, allowing the small patients plenty of fresh air, either at the seashore or in the mountains.

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HOMOLOGOUS CORNEAL TRANSPLANT.

ANCIL MARTIN, M. D.

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Report of a case of corneal transplantation in which the whole thickness of the cornea was replaced by the cornea from an eye about to be enucleated. Read before the meeting of the Arizona State Medical Association, April 25, 1918.

After reading the excellent history of corneal transplants and grafts presented by Dr. Sydney Walker, Jr., at the last meeting of the American Medical Association, I am instigated to present my personal experience and views on the subject of the homologous corneal transplant.

My work along this line has not been successful from a visual standpoint, but nevertheless it has some value in demonstrating the vitality of the eye structures. Furthermore, it presents some new points in operative technic, which may be of assistance to others who are experimenting with corneal transplant. I feel confident that ultimately thru operation by corneal transplant or graft, a successful method for restoring the vision of those blind from dense corneal macula will be evolved. We should be encouraged in our efforts when we consider that in the course of surgical evolution, a successful method for cure of many supposedly inoperable bodily defects has been demonstrated.

REPORT OF A CASE.

N. V.; Miner. Blasted April 4, 1915. Seen August 12, 1915. Left eye lost, phthisis bulbi. Right eye, a marked fullness of the conjunctival vessels, moderate ciliary injection and tenderness. Dense corneal leucoma. There remained a small area of cornea, about 2 mm. in width, near the upper sclero-corneal margin which was less dense, and thru which an indistinct view of the iris was had. Fine vessels covered the entire corneal surface. Tension —1. Pupil undoubtedly occluded.

Again seen April 16, 1917. Vision in the right eye, light perception only. A small area of the superior portion of the cornea, near the limbus, was relatively clear; and at that locality an iridectomy

was performed. The result was perception of hand movements, when the upper lid was elevated with the finger. This amount of vision being of no value, a corneal transplantation was performed, under the following technic.

By means of a hypodermic syringe the conjunctiva of the globe was ballooned with normal salt solution, and a division of it was made at its sclerocorneal insertion. The pouchlike conjunctival sac could then easily be drawn together over the cornea. Three mattress sutures were placed but not tied. An instrument having the diameter of 8 mm. was used to trephine the cornea. The degenerated iris was found adherent to the lens capsule, the lens cortex having been absorbed. The capsule, together with the remains of the iris, was gently drawn forward and excised with scissors, which procedure exposed the vitreous.

Using the same trephine, a corneal transplant was removed from the eye of another patient, whose eye was to be enucleated because of the presence of a rock within the globe; a negative Wassermann having first been demonstrated. The transplant was transferred to the eye of the first patient, where it readily adapted itself to its new position. No corneal sutures were used. The conjunctival sutures were then tied, burying the transplant within the conjunctival sac.

On the seventh day the eye was dressed. The sutures had cut loose and the conjunctiva was retracted, exposing the new cornea. With the nourishment furnished by the conjunctival flap, the transplant was alive and continued so, although it was found to be opaque. After two months the corneal opacity was somewhat cleared, so that the patient was able to discern hand movements. Tension was normal. At this time he returned to Old Mexico.

On the 20th of April, 1918, I received a communication from the chief surgeon of the mining company, in whose employ this patient had been at the time of the receipt of his injury, stating that "the corneal transplant is translucent and patient has slight perception of light."

REMARKS.

The features of this operation were the large size of the transplant. The method of dissecting the conjunctiva by ballooning, in its use as a support to hold the transplant in position. The use of the conjunctiva to quickly close the annular wound, and to aid in nourishing the transplant during the first week. Finally the removal of the iris and the lens capsule.

It has been the history of the major number of corneal transplants that opacity has supervened. This, without doubt, is due to the entrance of the aqueous into the interstices of the laminated corneal structure; causing a swelling of the tissues. Although the corneal laminae are more or less continuous with those of the sclera, nevertheless the thought suggests itself that a trephine large enough to divide the eye capsule just without the true corneal structure may pass thru tissues which will not readily admit fluids into the corneal parenchyma.

Working upon this idea, numerous operations were performed upon the cadaver. Another corneal trephine was secured, having a diameter of 13 mm., so arranged that the plunger can be set to limit the incision to any desired depth, and this should be set at a

little less than 2 mm. In its use the section is not always completed in the entire corneal circumference—in fact, it is usually necessary to raise the cornea gently and complete the excision by means of curved iris scissors, carefully following the line of incision made by the blade of the trephine.

The next step is the removal of the iris, which is accomplished by passing one blade of a flat forceps, beneath the iris, grasping it and with gentle traction, divulsing the iris from its origin, exposing the ciliary body and leaving the lens in place.

If the lens is found opaque, or becomes opaque following the operation, it is proposed that extraction may at a later period be made in the usual manner. The removal of the iris is done with the purpose of avoiding postoperative iritis and its consequences.

The transplant removed from the eye of the second patient, is placed in situ. The sutures, which have been previously introduced into the ballooned conjunctiva, as outlined in the description given in the case report in the early part of this article, are then gently tied, the conjunctiva thus covering the entire cornea.

The operation is very readily performed upon the cadaver, in which the ocular tension is decidedly minus; and should more easily be performed upon the live subject having normal tension. It is not expected that the result will produce a perfect visual organ. Should the cornea remain clear, as we hope it may, some degree of useful vision would follow.

IRIDECTOMY FOR OPTICAL PURPOSES.

J. H. McKELLAR, M. D.

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A discussion of the indications and technic of optical iridectomy with report of two favorable cases.

Iridectomy is performed for one of two purposes, either as a therapeutic measure, or as an optical measure to improve sight. The latter, or optical

iridectomy, is considered in this short paper. The operation consists in removing a piece of iris in such a position that rays of light are admitted

thru clear media. It is usually done because the path of light which normally passes thru the pupil is obstructed as the result of a disease process, that is, when there is an opacity of one of the media and that opacity is in the pupillary area.

INDICATIONS.—1. Any obstruction to the before mentioned path of the light thru the pupil, which is permanent in character, and of such extent as to render the eye unserviceable for vision; in an eye where such obstruction may be avoided by removing a section of iris, giving a new path for the rays of light entering the eye. It should only be done if the vision is decidedly reduced as a result of the obstruction.

The most usual of these obstructions are leucoma, the result of corneal ulcer, or interstitial inflammation of the cornea; pupillary membrane following iritis or iridocyclitis; or central cataract. The eye should be free from inflammation, as otherwise, especially in pupillary membrane, the opening is apt to fill with exudate after the operation. For pupillary membrane, it is well to postpone the operation for at least a year from the time the eye becomes quiet.

2. Rarely in marked displacement of the pupil, due to incarceration of the iris.

These indications only hold good if the vitreous is presumably clear, and if the retina and optic nerve are in good condition. This may be determined by testing the patient's light perception, and light projection. When only quantitative vision is present, the test may be made by means of a lighted candle in a darkened room. The patient should be able to point out the position of the candle readily, as the most dense opacity of cornea or lens will not cause loss of ability to locate a source of light. The limits of the visual field may also be determined with the candle, and should be normal, or nearly so in extent. The amount of vision should correspond approximately to the degree and position of the obstruction.

CONTRAINDICATIONS.—1. When light perception, and light projection are deficient, or absent; also when the field

of vision is much contracted without increased tension.

2. Where the eye is abnormally soft, or small, or the cornea flat, as these conditions usually indicate diseased conditions back of lens.

3. Strabismus which has lasted for a considerable number of years in the eye in which the opacity is present. In such a case amblyopia ex anopsia is almost surely present; and the result will be probably disappointing, tho the operation might be tried if the case be otherwise visually hopeless.

4. Where no reasonably clear path for the entering rays can be secured.

5. When inflammation of the eye exists.

OPERATION.—The operation does not differ materially from the therapeutic iridectomy, excepting that the portion of the iris removed is usually narrower, and does not extend back to the root of the iris. To make such a coloboma the section should be short and not extend back of the limbus. Such an opening limits the dazzling, and also the irregular refraction, and prismatic action of the margin of the lens. The position of the iridectomy in opacity of the cornea must depend upon the area of the cornea most transparent. If the opacity is central, and the periphery is all equally clear, my own choice is upward, altho this seems to be contrary to the teaching of authorities, as in this position the deformity is least apparent, and the amount of light entering the eye can be readily regulated by the action of the upper lid. If the iris is adherent to the lens capsule one must be exceedingly careful in stripping it off with the iris forceps, not to injure the lens. In making the section the keratome must incise the cornea almost at right angles, never obliquely splitting the corneal layers, as in this case a new opacity is apt to be formed over the coloboma.

RESULTS.—Too much should not be promised to the patient for, even tho an unobstructed path be secured for the rays entering the eye thru the eccentric pupil, there is almost certain to be a marked astigmatism, more or less irregular, especially if the iridec-

tomy is placed in the extreme periphery. Yet in carefully selected cases, the visual result is decidedly gratifying.

The prognosis as to vision depends upon the location and density of the opacity, and the general condition of the eye, especially upon the functional condition of the retina, and optic nerve. The intraocular tension must also be considered. The visual result is apt to be better if the obstruction in the pupillary area is very dense, and strictly localized; and in a central scar of the cornea, tattooing may obviate some of the dazzling after the operation. If there is a pupillary membrane due to an old iridocyclitis, the result may be disappointing. The segment of iris may be removed, but the pigment layer will sometimes remain adherent to the lens. The deformity owing to the defect in the iris is not great, and if above is scarcely noticeable. Some times dilating the pupil to its maximum, if it is not bound down, will give some indication as to what may be expected as a result of the operation. If the pupil is secloded, and occluded, and secondary glaucoma is present, the iridectomy may relieve the increased tension in addition to its optical effect.

The line of vision is not changed unless the new pupil is very peripheral, in which case the prismatic action of the edge of the lens may displace the visual field downward.

CASES.

Case 1.—Mr. J. F. L., age 51. Had an operation on his left eye when 24 years of age. Right eye became affected two years afterward, presumably by sympathetic inflammation. Since 29 years of age he has been practically blind. Examination: Patient healthy. No history of tuberculosis or

syphilis. Wassermann negative. Right eye. Lids normal. Pupil, nearly secloded and occluded, central lenticular opacity apparently. Tension normal. Vision: Can distinguish outlines of large objects in a good light with great difficulty. Practically little more than light perception.

Left eye: Phthisis bulbi.

Operation: Iridectomy, above, in right eye. Iris adherent to lens except at periphery. Iris pigment remained adherent to lens except at periphery.

Result: Patient has astigmatism of about 7 diopters, sees to get about, and do some work. Can read large type, and tell the time by his watch. In this case the iridectomy was of necessity very peripheral, and the visual field is projected downward unless the patient wears a strong prism base up.

CASE 2.—Mrs. S. E. J. Age 44. Vision has been very poor since her earliest recollection. Of late years the condition has been worse. Examination: Patient a deaf mute, healthy, has no tuberculosis or specific history. Lids healthy, dense scars of each cornea in pupillary area. Light perception and projection good in each eye. Pupils in each eye apparently secloded. Vision: Right eye, light perception.

Left eye, fingers with difficulty at four feet.

Operation: Optical iridectomy upward in both eyes.

Result: Much better in right eye, as the iris in left eye was adherent to the lens, and the segment could not be cleanly removed. Patient has an astigmatism of about five diopters.

Distance vision about 20/40. Can read the finest type with or without glasses, and carries on a considerable correspondence with her friends.

Both of these patients have been restored from practical blindness to a condition of having serviceable vision.

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APPARENT ACCOMMODATION IN APHAKIA.

WILLIAM ZENTMAYER, M. D.

PHILADELPHIA, PA.

Report of a case of such apparent accommodation, with discussion of the possible conditions that might account for it. Read before the Section on Ophthalmology of the College of Physicians of Philadelphia, March 21, 1918.

More than a century ago Thomas Young made investigations as to the existence of accommodative power in the aphakic eye and wrote that the results obtained, although inconclusive, indicated an absence of that function. Later von Graefe asserted that some accommodative power remained, but recognized that those who made the most accurate, and on repeated investigations, the most uniform statements, had the least range.

Donders¹ then took up the problem and tried to solve it by observing whether a change took place in the form of a distant point of light during convergence and concluded "that in aphakia not the slightest trace of accommodative power remains."

While these studies determined that accommodative power in the ordinary meaning of the term does not exist in eyes deprived of the crystalline lens, the fact remains that aphakic eyes may, and frequently do, have the inherent power of reading at varying distances with or without the lens which adapts the eye to parallel rays of light.

Literature contains the recital of a number of such cases and from the varying conditions which have been present numerous solutions of the problem have been offered.

Fuerst² found in 8 cases out of 20 of juvenile aphakic eyes, after congenital cataract or cataract acquired in the first years of life, a more or less considerable accommodative power present so that sometimes the entire amount of high grade H. of the emmetropic aphakic eyes (12-13 D.) for distance as well as near was compensated for.

Pfalz's³ case of congenital dislocation of the lens had an apparent accommodation of 4 D.

In Landrieu's⁴ case of aphakia in a boy of 9 years, operated upon. V. with $+15 D = 5/6$ and type could be read at the usual reading distance.

Collins's⁵ case was a boy who was operated on at the age of 7 by repeated dissections, and at the age of 14 could read Jr. 1. fluently with his distance glass, $+13 D. S.$

CASE.

My own case is that of a girl 8 years of age with acquired cataract. Two needlings were fruitless in securing absorption, altho a bud of cortex protruded thru an opening in the capsule, so that for the third and final operation a broad needle was introduced thru the cornea, at the site of the capsular rent, and immediately vitreous came thru into the chamber, leaving a clear opening about 2 mm. long and 1 mm. wide surrounded by opaque lens matter. Thru this opening she has $5/5 V.$ and reads 0.5 M. fluently to within 3 cm. of the cornea.

From painstaking investigations and from pure theorizing the following explanations have been evolved for this interesting and fortunate result.

1. That it is due to corneal astigmatism.
2. Increase in the index of the refractive media.
3. Partial regeneration of the lens, the functioning elements being forced into the pupillary space thru external muscle pressure. (Fuerst.)
4. From cutting off the circles of diffusion either by contraction of the pupil, by nipping of the lids or by a stenopaic opening in the secondary cataract.
5. Lengthening of the optic axis by contraction of the external muscles.

6. Forward bulging of the anterior surface of the vitreous, or an increase in corneal curvature by pressure of the external muscles.

7. Spherical aberration (largely compensated for by the lens in the normal condition) (Kröner).⁶

8. Thru adjustment of the cataract lens.

It is probably true, as Donder's asserts, that many who have written upon the subject have had no idea of the degree of distinctness of vision even in imperfect accommodation and that many of the cases of apparent accommodation may thus be explained.

Schmidt-Rimpler⁷ states that with his own aphakic eye on close observation of single letters he finds vision to be perfectly sharp only at a single distance. I believe that many are unaware of the ability of an eye without accommodation to read without a glass very fine print up to within a few cm. of the cornea, thru a pin hole aperture in a card held close to the cornea (of course we are all aware of the fact that an uncorrected highly astigmatic eye can have normal vision thru a pin

hole). This ability to read by cutting down the circles of diffusion I believe explains my own case as Collins and also Landrieu explain their cases.

Other contributing factors are, no doubt, irregular astigmatism and also regular astigmatism, tho Kröner found that the region of accommodation does not correspond to the optical distances between the two foci; and that it is generally greater in eyes with little astigmatism and less in highly astigmatic eyes.

This author believes that in general it results from influences which by diminished visual acuity lessens the necessity of exact focussing. Fuerst believes it to be caused by a vicarious function from the pressure of the orbicularis and external muscles which develops sometimes after the operation and only when the correcting lenses have been withheld from the patient (this is disproved by Collins' case and also by my own), and further believes that it finds a certain analogue in isolated observations of abnormal refraction in eyes containing their crystalline lens.

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NOTES, CASES, INSTRUMENTS

OPHTHALMOPLEGIA AND OPTIC NEURITIS.

Report of Cases.

FRANK A. MORRISON.

INDIANAPOLIS, IND.

OPHTHALMOPLEGIA FOLLOWING PROPHYLACTIC DOSE OF ANTITOXIN.

Boy, age 7. History as given by the mother and grandmother. Last Sep-

tember one of the other children of the family was attacked by diphtheria. This boy was given a prophylactic injection of antitoxin. Three days later his eyelids began to droop, and have continued to do so up to this time, altho the mother thought at times they were better.

The child presented the ordinary picture of complete ophthalmoplegia. The mother persisted in the statement that

the child had never been sick and that nothing peculiar in its gait in walking, speech or swallowing had ever been noticed. Unfortunately it was not possible to communicate with the physician in attendance and the amount of antitoxin injected could not be learned. Diagnosis: uncertain, ophthalmoplegia possibly from antitoxin or possibly from a mild and unrecognized diphtheria or even the possibility of a mere coincidence must not be overlooked.

OPTIC NEURITIS WITH RECOVERY.

Girl twelve years of age. Had been perfectly well, but noticed one morning about a week before coming to my office that she could not see the largest writing on the blackboard at school, with her right eye, even when a few inches away. She was positive she could see when she went home the night before. This child was an unusually bright girl and mentioned several experiments which she had made in the way of testing her vision a few days before she discovered her condition. Her home surroundings had not been good and she had been subjected to a great deal of distress owing to the misconduct of her father. She had no headache, vomiting nor indeed symptoms of any kind. Reflexes all normal. No indication of paralysis or lack of coordination. Pupils responded to light and accommodation normally. Fundus of right eye showed typical choked disc of three diopters with a few retinal hemorrhages. Further questioning as to the possibility of injury brought out the fact that she had fallen upon her back a few days before the appearance of the eye symptoms; but she insisted she had not been hurt. Being pressed to tell just how she struck when she slipped she finally said, "Why I never thought of it but I don't believe I remember. Do you think I could have jarred my senses away?" There was no bruise upon the head or spine to indicate injury. She returned home in a neighboring town with a rather uncertain diagnosis relative to the cause of her condition and a still more uncertain prognosis.

A few weeks later she was sent to a hospital in this city for observation and again came under my notice. An examination of the fundus showed the same condition as when last seen but somewhat less marked. She was selected as one of the patients to have a mydriatic put into the eyes for class demonstration four days later. When she came before the class at the end of this period the fundus was almost normal and the improvement continued to complete recovery. While in the hospital X-ray pictures of the head showed in the language of the resident surgeon "a faint shadow in the occipital region which might indicate a slight fracture."

CASE OF HOLE IN DISC.

CHARLES B. HARWOOD, M. D.

HOUSTON, TEXAS.

The patient, Mr. K., aged 28, had vision in the right eye of 20/200. Ophthalmoscopic examination of the right

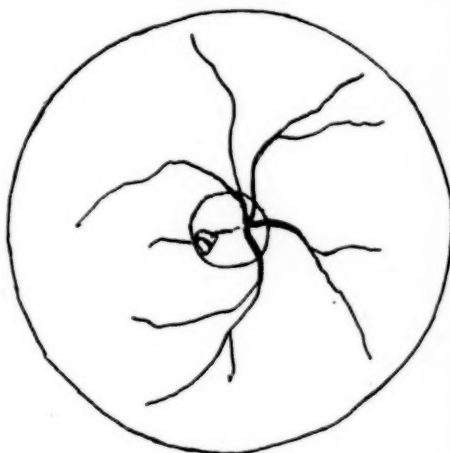


FIG. 1.

Harwood's case of hole in disc. Diagram showing vessel at bottom of hole near temporal margin of disc.

eye showed a triangular hole on the temporal side of the disc just below the center. The edges of the hole overhung the floor as shown by two minute blood vessels which disappeared at its edge. A -8 D. lens showed another blood vessel at the bottom of the hole crossing it diagonally.

REPORT OF A CASE OF BILATERAL LUXATION OF THE LENS.

FRANK ALLPORT, M. D., AND JAS.

SMITH, M. D.

CHICAGO.

Cases of this sort are always of great interest on account of the infrequency of their occurrence, hence we shall report this case as somewhat of an ophthalmologic curiosity.

According to Fuchs, changes in the position of the lens always have their anatomic cause in changes of the zonula of Zinn—either elongation, rupture or destruction of the zonula, with a subsequent displacement of the lens.

The patient, age 5, had enlarged eyeballs. The eyes suggested the buph-

thalmic type, altho the tension was not raised. There was no tremor of the iris. A mydriatic was instilled in order to make a fundus examination, and also estimate any error of refraction by means of retinoscopy. When the pupils were dilated, both lenses, entirely loose and unattached, and which were clear, floated gracefully into the anterior chamber, and upon the child changing the position of its head, they would float to various locations in accordance with the position of the head, even back into the lens fossa. Luxation of the lens was not suspected at the preliminary examination, and indeed was not evident until mydriasis was induced. The child was entirely unmanageable—had this not been the case, the luxation of the lens might have been manifested.

SOCIETY PROCEEDINGS

ROYAL SOCIETY OF MEDICINE. SECTION ON OPHTHALMOLOGY.

June 12th.

President, MR. WILLIAM LANG, F. R. C. S.

Before entering upon the agenda, the President referred in sympathetic terms to the recent decease of Mr. John Couper, an old colleague of his at the great City Ophthalmic Hospital in City Road, always known as "Moorfields." The chief claim of Mr. Couper to fame would be his special ophthalmoscope. Couper's direct method of ophthalmoscopy made possible the minute observations associated with the late Marcus Gunn, a work since perpetuated by Mr. Bardsley.

Cyst in Front of Disc.

Dr. G. H. Goldsmith exhibited a case of cystic swelling of the disc. On the left side was a large coloboma of the iris and of the choroid. The disc had suffered almost complete eclipse, by a large swelling which he took to be cystic. It was semitranslucent and

spheroidal, and over the swelling ran two vessels.

Mooren's Ulcer of Cornea.

Mr. F. A. C. Tyrrell showed a patient with Mooren's ulcer. It showed very extensive denudation. The patient attended St. Mary's Hospital about a year ago, with a large denuded area, and but a small area of epithelium in the center of the cornea, about 4 mm. across. Two attempts at cauterizing did not bring about healing. He therefore tried the conjunctival flap method, using purse-string sutures. The conjunctiva adhered to the denuded area in the outer part, and the cornea vascularized, but the case did not progress very satisfactorily. After keeping the patient under observation in the hospital a month, he decided to unite the lids in the center, so as to be able to flush the conjunctival cavity thru from side to side. The cornea was now clearing up, and the patient could see fingers at about a meter distance; also the pupil and iris could be seen. He showed it as a sort of sequel

to the similar case he exhibited about a year ago.

DISCUSSION.—The President said he touched the cornea in a case of ulcer with pure flavin. The patient said it was very painful, but healing occurred. Many cases did well with weak solutions: this was the only case in which he used the material pure.

Mr. Leslie Paton said he had been watching this case shown by Mr. Tyrrell, and when she came for treatment the area of cornea which seemed intact was steadily becoming smaller: moreover, the area left was insensitive. It was the experience gained in connection with neuropathic keratitis which led to the sewing of the lids in this case. Clearing up of the cornea followed this measure equally well in herpes in which there was deep corneal ulceration. But in the keratitis following removal of the Gasserian ganglion the case seemed different because an attempt subsequently to unsew the lids led to a relapse: that had been his experience. There must be left at least a bridge of tissue of not less than one millimeter.

Mr. J. B. Lawford said his experience differed from Mr. Paton's in this regard. Tho he had sewn the lids in a number of cases of operation on the Gasserian ganglion done by a colleague, in none of them was separation of adhesions later followed by relapse.

Choroidal Scar.

Mr. M. L. HEPBURN showed a drawing of a case with an old choroidal scar with a fibrous band stretching from it to the disc. The patient came for glasses in the ordinary way, and was not aware of any defect. Vision was 6/9. Such a condition was common after accident or inflammation; but here there was no history of either. Possibly it was a congenital condition.

Cyst of Iris After Injury.

Mr. A. ROXBURGH showed an implantation cyst. The patient, a girl, aet. 14 years, had her eye struck by a tin toy aeroplane. The mother stated that the eye was operated upon immediately afterwards, probably the prolapsed iris was removed. A fortnight ago the girl complained of some pain,

pricking, and headache. The mother then noticed a dark spot in front of the eye. Vision had fallen from 6/6 to 6/12: tension was but little raised. In the left cornea was some iris adherent to the back of the cornea. Beneath the cornea was a translucent cyst, about $4\frac{1}{2}$ mm. wide, which appeared to be embedded in the iris, looking in every respect like an implantation cyst. It was increasing in size. He felt it his duty to tackle it, but he had never operated upon an implantation cyst of the iris.

DISCUSSION.—Mr. Treacher Collins agreed with the diagnosis, and said it was very important that the cyst should be removed. Otherwise it would certainly fill the anterior chamber, and cause increased intraocular tension. In removing it, it was essential to include the portion of iris which formed the back of the growth. If that bit of iris were left, the growth was practically sure to extend.

The President agreed with Mr. Collins, adding that the extent of these cysts often proved to be greater than it appeared to be.

Luminous Test Type.

Mr. M. S. MAYOU showed, for CAPT. MOXON, test types of a luminous character, on the principle of the luminous wristwatch, for estimating the degree of light sense and night blindness. After getting accustomed to the darkness, a normal person could read these types in a completely darkened room at a distance of 4 feet. Mr. Mayou had ascertained that the paint used was composed of radium bromid and zinc sulphid, the light being due to the radium rays bombarding the zinc sulphid. It could scarcely be regarded as a standard, however, seeing that the luminosity decreased with the age of the composition, and bore a relation to the thickness of the lettering. The scientific standard would be a screen illuminated from behind, the observer wearing green glasses.

Neuroretinitis with Chickenpox.

Mr. LESLIE PATON showed a patient in whom chickenpox was associated

with neuroretinitis. There was only one other case of the kind on record, tho neuroretinitis had been recorded in connection with polioencephalitis. The present patient had had nothing else amiss with him, and his blindness came on suddenly on the third day of the chickenpox. The large central scotoma was now gradually clearing up.

Bifocal for Myopes.

MR. P. C. BARDSLEY read a communication on a new form of bifocal for myopes. He said there had arisen a school of ophthalmologists who taught that in early life myopia should be fully corrected, the subject continuously wearing glasses to fully correct, however high the degree of defect might be. This school argued that high correction only turned the child or young adult with myopia into a person with normal vision, and that thereby the accommodation was called into play, as was the accommodation of the emmetrope or the hypermetrope. The same people stated that if this full correction was not given, certain dire results would ensue: accommodative power would be lost, and rapidly progressive myopia would take place. But no evidence had been adduced that lack of full correction of myopia resulted in an increase of myopia.

The author had himself been myopic since early childhood, and it had but little increased since. He had objected to prolonged study in glasses which gave full correction, and when well over 40 years of age his accommodation was found to be more powerful than any recorded up to date at the Oxford Ophthalmological Congress. Why, he asked, should myopes be robbed of the privilege of reading without using their ciliary muscles?

On looking down thru the lower glass in the ordinary bifocals, there was a considerable distortion of the image. To overcome this, the author angled his bifocals. They were made of separate pieces of glass slightly beveled against one another where the pieces met. Next, he angled the circular bifocals, and to overcome the defect of bringing the edges of the upper

and lower glass into continuous contact, he ground a toric curve on the upper lens, and a double concave or convex on the lower lens. He detailed the advantages resulting from this improved kind of spectacles.

Hysteric Disorders of Vision.

MR. L. R. YEALLAND read a paper on hysteric disorders of vision.

The paper was based upon Dr. Yealland's experiences as resident medical officer at the National Hospital for the Paralysed, Queen Square. He said hysteric disorders of vision could be grouped, clinically, into two classes: (1) conditions in which contraction of antagonists could be demonstrated, (2) those in which this could not be demonstrated. By contraction of an antagonistic group of muscles he meant the contraction of a group which should be relaxed during the performance of a given movement. This could be demonstrated in blepharospasm, ptosis, and spasm of accommodation. In blepharospasm there was simultaneous contraction of the levator palpebrae superioris and of the orbicularis oculi. Failure of relaxation of the last-named muscle prevented the opening of the eye, hence there was apparent loss of vision.

The action of the antagonists in spasm of accommodation could be explained on the assumption that the suspensory ligament of the lens performed an opposite action to that performed by the ciliary muscle. When the latter contracted, the ligament relaxed; when the ligament was in a state of tension, the ciliary muscle relaxed. The phenomenon of contraction of antagonists could not be demonstrated in such conditions as limitation of the visual fields and amblyopia, but in these conditions, contraction of antagonists in some other part of the body could be observed. The treatment was one of suggestion, and must be got thru at one sitting. It produced complete relief of the physical disability, tho much could not be hoped for in improvement of the mental state.

H. DICKINSON.

CHICAGO OPHTHALMOLOGICAL SOCIETY.

April 15th, 1918.

President, DR. HEMAN H. BROWN.

Convergent Strabismus Treated by Atropin and Glasses, with Some Cases of Hereditary Strabismus.

DR. CLARENCE LOEB read a paper on this subject which will be published in full in this JOURNAL.

The Surgical Treatment of Strabismus.

DR. H. W. WOODRUFF stated that the surgical treatment of strabismus must be considered when other methods of treatment have failed. In most instances the object to be obtained is cosmetic rather than an improvement in function; altho the latter is occasionally obtained when the visual lines have been approximated by a successful operation.

The author has been doing the tucking operation for many years, in preference to advancement; usually combined with partial or complete tenotomy of the opposing muscle. This operation has been done without the use of the tendon tucker. Briggs, he said, has apparently simplified the tucking operation by his method of clamping a wire loop over the folded tendon, capsule and conjunctiva. It occurred to the speaker that if this operation would exclude the conjunctiva, and catgut sutures were used in place of wire, it would be ideal. He has not as yet completely satisfied himself that the older tucking operation which he has been using for many years should be given up. However, the simplicity of the new one recommends it.

After describing the tucking operation, the essayist drew the following conclusions: 1. Operate at any age when local anesthesia is permissible, and nonsurgical measures a failure. 2. Never do an advancement, but always a tucking in any degree of actual concomitant strabismus. 3. Tenotomize the opposing muscle, when necessary, to increase the effect of the tucking. 4. In convergent strabismus, never secure an over effect when tenotomy is

used. If such a result is accidentally obtained, correct at once by a suture. 5. When complete tenotomy is performed, guard against the sinking of the caruncle by using a conjunctival suture. 6. In paralytic strabismus the operation of tendon transplantation should be used.

DISCUSSION.—Dr. Robert von der Heydt stated that there are more eyes lost to usefulness from neglected squint than by all other causes. All squinters have a hereditary taint in the form of a muscle imbalance. This, together with reduced visual acuity in the one eye, from any cause, or high hypermetropia will bring on a squint. Alternating strabismus is so marked a muscle imbalance that it overcomes the fusion tendency; or the angle may be such that the object looked at falls on the blind spot of the squinting eye, hence monocular suppression is more easily acquired. Refraction under a cycloplegic is the first step in the treatment of squint. In case of failure to create parallelism by glasses, atropin in the fixing eye may be used. If we can by its use transfer the work to the squinting eye, we may educate this eye to fixation, later fusion and permanent parallelism. If not, occlusion must be resorted to. A method of handling this variety of squint he presented at the meeting of the Society last October.

Dr. D. T. Vail, of Cincinnati, Ohio, stated that the teachings of Valk conformed so closely to his own ideas and observations that he feels there has been no real contribution to the etiology of strabismus since Valk's publication some ten years ago. He believes that Valk stated that so-called amblyopia ex anopsia is one thing and congenital amblyopia is another. We may have congenital amblyopia with no strabismus. He has seen many cases where the affected eye was perfectly straight and had only 20/200 vision and could not be improved with any amount of atropin, training or glasses. The same is true where the congenital amblyopia is associated with strabismus.

Nothing improves the vision in true congenital amblyopia. Amblyopia ex

anopsia associated with convergence is entirely different. Here we have an amblyopia because the child has an ex anopsic eye which is possessed of a high degree of ametropia, and the visual image in it is suppressed. Usually there is compound hypermetropia, and the convergence is caused by the excessive action of the internal rectus as-sociation with accommodation effort. In such cases one may almost invariably accomplish a good deal by glasses, atropin and training.

There are many different types of convergent strabismus, and each case must be studied on its own merits and a differential diagnosis made. He has seen quite a few cases with double paralysis of the sixth nerve in which there was inveterate double convergence. In these cases the refraction and vision in each eye may be normal, and yet the convergence is extreme, with no power in either eye to bring the axis of vision beyond the median line. Such patients will use the right eye for seeing objects in the left field, and the left eye for objects in the right field. In spite of everything one may do, these patients will continue to "cross fire."

Referring again to the subject of congenital amblyopia, the speaker called attention to the excellent book of Collins and Mayou on "Pathology and Bacteriology of the Eye," stating that this book contains an explanation of what congenital amblyopia is, why it is, and why no amount of treatment or operation succeeds in improving it. Collins, he says, teaches that there is a lack of differentiation of the macula lutea in congenital amblyopia. By this he means that the macula lutea is not particularly different from other parts of the retina. There is no macula lutea. If one will examine the region of the macula in a case of congenital amblyopia ophthalmoscopically, and compare its appearance with that of the seeing eye, he can satisfy himself that this teaching of Collins is correct.

When about to undertake an operation for concomitant squint on little folks four or five years of age, the speaker sometimes has been surprised to note that the condition has entirely

disappeared under the anesthetic. He has seen cases in which the eye that was convergent became markedly divergent under the anesthetic. During sleep such eyes must be divergent and during waking hours convergent. He has tenotomized cautiously such cases with success, and in some of them has noted in after years a well developed divergence. On the other hand, he has seen such cases that after ten or more years have perfectly straight eyes with normal fusion power.

Another class at close range can fuse very nicely and hold the focus, but the moment the accommodation relaxes, as when looking at infinity, the poorer eye becomes divergent. He thinks it is best to use the plan of atropin and glasses when there is a strabismus of less than 15° . When it is more than 15° , he recommends tenotomy. A simple complete tenotomy would insure 15° of correction, but not more than that.

As regards tucking, he thinks it is only a temporary affair. The temporary help which it gives is very great, and it really amounts to a permanent cure in many cases. But the tuck unfolds in 10 days or a few weeks and is no longer a tuck. In order to accomplish any good with a tuck one must cut or weaken the opposing muscle. The speaker has accomplished just as good results by putting in a reef stitch as by the tucking procedure, but a reef stitch will not hold as long as a tuck; it pulls out sooner. The reef stitch is used for divergent strabismus where one cannot get too much effect and is not worrying about convergence following. He puts a strong stitch in the sclero-corneal tissue and carries the needle deeply thru the substance of the caruncle, and then by tying the margin of the cornea flush with the caruncle, he can, in divergent strabismus, produce a marked temporary over correction which compels the severed tendon of the external rectus to retract, thus gaining the desired permanent result with no fear of permanent convergence. The stitch is removed on the third, fourth or fifth day.

Dr. Oscar Dodd thinks that the question of the treatment of strabismus can neither be considered purely operative nor as one in which atropin and glasses alone are used. Every case of strabismus, unless of adults who have had strabismus for years and where operation is indicated simply for cosmetic reasons, should be gone into very thoroughly and studied under atropin and correction with glasses, to see how much effect can be gained. Most of the children with strabismus who come to him are amblyopic to quite a large degree in the converging eye.

If one eye is practically normal and the other one has less than 20/40 vision, the use of atropin and the correction of the hypermetropia and astigmatism with glasses will not effect a cure. In most of those cases one has only begun treatment. When the patients are young, six, eight or under ten years, he finds that it is necessary to keep the good eye covered constantly, sometimes for several weeks to correct the amblyopia. When that is done, he has seen the vision improve, from 20/100 to 20/40 in a few weeks. Using the amblyopic eye alone for one or two hours daily is absolutely ineffective. Educational treatment and development of the fusion sense with the amblyoscope is of value, but it is difficult to use with children.

If the amount of strabismus is 10°, 15° or 20° after securing all the benefit possible from the use of glasses, he believes operation should be done. He is not successful in operating on children under local anesthesia, as it is very rarely that they will submit to the operation which he deems necessary. He would not do a tenotomy on a child, expecting it to be permanent, because he has had patients return after 10, 15 or 20 years with divergence, when the result of the operation seemed absolutely perfect at first. Nothing should be done in these cases but tucking and advancement. In the tucking operation he puts his stitches thru the insertion of the tendon so as to get a good fixation point, using catgut which absorbs in from 10 to 20 days. Doing a

tucking operation with the instrument Dr. Woodruff has demonstrated is certainly a very easy and quick method.

Dr. Colburn stated that he devised a muscle tucking operation at about the time when the American Medical Association met in Columbus, 1899, and made a preliminary report on it. An instrument maker made for him a muscle tucker at that time. He did not follow up the use of the tucking operation because he found that he had a rather annoying puckering, so that after two or three years of use he abandoned it almost entirely, excepting as an aid to general tenotomy, tucking on the opposite side. The various means of operating, whether to tuck or advance, are debatable. One should take into consideration not only the condition of the refraction, but the anatomical condition of the eyes and head.

Dr. William A. Mann stated that when a patient first comes in for examination, if the convergent eye will not fix and stay fixed, he has very little hope of doing anything with atropin, if the vision shown is 20/200 or less. In these cases, if one will cover the good eye, the other eye will wander. On examination one finds not only an absence of the macula lutea but an increased number of blood vessels in the macular region. One may find the blood vessels almost running thru where the macula should be. In those cases of congenital amblyopia glasses are practically no good. He does not recall a single case where he has succeeded in developing central vision.

Dr. von der Heydt, in connection with the discussion of tucking methods, exhibited a little silver clamp he has designed for advancing a muscle. The muscle to be shortened is drawn thru the clamp and held by two screws. It is then left in the eye until the strangulated muscle sloughs off. He has used it in three cases thus far with success. It took eleven, twelve and fourteen days respectively. Some of the clamps were passed around for inspection.

Dr. Clarence Loeb, in closing on his part, stated that he did not agree with

Dr. von der Heydt in regard to the success in alternating strabismus. Those cases, as a rule, have been most successful under the treatment described. As to Dr. Vail's statement about paralytic squint, one could not expect any improvement from the use of glasses, but he could not expect it, on the other hand, from the operative side. The report was rather an attack upon the theory that all cases should be operated, rather than to insist that all cases would be cured by the use of glasses and treatment.

As to the question of differentiating between amblyopia ex anopsia and congenital amblyopia, the speaker does not think it is quite as simple as indicated. He has examined quite a number of young children with convergent strabismus, and although one eye had less vision than the other, he could not recall that there was any difference in the macula. The question of strabismus and amblyopia ex anopsia is not that the strabismus results from the amblyopia, but that the strabismus causes it. If this were not so, he could not explain the experiences of the last couple of months in connection with advisory boards, where they have had quite a number of patients whose vision in one eye was decidedly below the other, and at the same time there was no strabismus. If amblyopia causes strabismus there should be a much higher degree of strabismus than found.

Dr. Woodruff, in closing, said that in paralytic strabismus the operation of tendon transplantation should be used. The speaker has seen and has operated on two cases of paralysis of the external rectus muscle. The etiology in one case was possibly traumatic, a head injury; at least, that was the only history obtainable, while the other was probably a congenital case. It was his first experience with muscle transplantation, and the result was much more satisfactory than with any other operation which he had ever attempted. In a paralysis of the external rectus, parallelism can be maintained looking straight ahead, but with very little ability to rotate the eye out-

ward. That was his experience in these cases, but one of these cases gave an exceedingly gratifying result. This case was described in detail.

Unilateral Papilledema, Case for Diagnosis.

DR. HUGHES, of Elgin, reported the following case:

Mrs. J. R., aged 22. Referred by Dr. R. on March 25, 1918, who asked for a fundus examination. Patient's mother died seven years ago at the age of 39, from uremic poisoning subsequent to the removal of the kidney for tumor. Mother was subject to severe vomiting spells and headache which lasted several days at a time.

Personal history: Patient has had most of the diseases of infancy. Typhoid fever at 10 years. Was poisoned from milk when several weeks old and has been sickly since. In 1913 she was ill for several months with what a physician called the early stage of tuberculosis in the left lung. She spent several months in the country and apparently recovered. She had scarlet fever in 1915.

Present history: Has been subject to headaches for several years. Last July she had a very severe headache accompanied by nausea and vomiting, dizziness, and a tendency to fall to the right side. There was very severe noise in the head and right ear at that time. At first, the headache came at intervals of one month, at the time of menstruation, but later became semi-monthly, and at present weekly or more often. Patient states that after the attack she feels like a man "after a jag." There is a tendency to fall to the right for several days, and lying on the right side causes a feeling of dizziness and falling to the right. Headaches are frontal and fronto-occipital. Of late the menstrual flow is getting less. She is deaf in the right ear, is losing weight constantly. She has been married for six months, but no pregnancy. No loss of vision in an attack, but there is marked increase in the head noise.

Patient looks worn and rather anemic. Nose shows a septal deflection to

the left side with congestion of the inferior turbinate. Sinuses negative. Throat negative. Right ear drum retracted but mobile; reflex still present; no congestion. Left ear negative. Hearing: Galton all the way, both ears. Voice, right ear loud voice at 14 feet, whisper at 8 feet. Left ear normal. Turning to the right produces dizziness with a tendency to fall to the right. No nystagmus. To left normal. Tubes normal in both ears. Inflation does not improve hearing.

The appearance of the eyes is normal. Tension normal. Reaction normal to light and accommodation. Vision of 20/20 in each eye. Fundi: right eye normal; left eye choked disc of about two diopters. Fields: right eye normal; left eye contraction of form field to 35 above, 30 nasal, 60 temporal, 30 below. Concentric contraction of color fields. No active specific infection, but reaction sufficient to say an inherited type of infection. Blood pressure: Systolic, 130, and diastolic, 100.

Dr. Hughes stated that his object in reporting this case was to get some information as to the nature of the lesion and where it is located.

DISCUSSION.—Dr. Robert H. Good stated that the case appeared to be one of intracranial pressure. There was some edema of the right optic disc as well as the left. In addition there is involvement of the right ear. The patient became sick suddenly in July with severe headache, nausea and vomiting. This nausea still continues every time she has a recurrence of the headache. According to the patient, there is a peculiar numbness in the right arm and right leg. The speaker recommends lumbar puncture with a view to determining the pressure of the spinal fluid. If the pressure is shown to be increased, one knows then that there is an increased intracranial pressure.

The picture resembles to some extent that of cystic involvement of the temporal lobe of the right side of the brain. On pressing the head very hard the right temporal region is very tender. The patient complains very much from this pressure, so that the lesion is

undoubtedly located in the right temporal lobe of the brain. Wherever one finds marked tenderness in a certain area, it is usually a fairly good indication of the location of the trouble. He is absolutely of the opinion that if the lumbar puncture shows an increased intracranial pressure, this patient should have a decompression. That it is not a tumor, one may assume, from the fact that the attacks occur at intervals; and between intervals there is no headache and no pain. If there were a tumor present, one would expect the sight to be impaired by this time. It is probably a cystic condition of the right temporal lobe of the brain.

Dr. D. T. Vail, of Cincinnati, said the case of Dr. Hughes is one of interest on account of its obscurity. The history of headache and vomiting, associated with other symptoms, seems to clearly indicate excessive intracranial pressure. The case calls for a careful differential diagnosis, but the onset is so recent and the symptoms so indefinite, that about all one can now say certainly is that there is increased cerebral pressure, and that is a condition common to several distinct and widely differing brain maladies. The speaker is impressed with the thought that the present stage is transitory, and that later on characteristic symptoms will establish themselves so that a definite diagnosis will then be possible.

In differentiating one should first endeavor to learn whether the lesion is anterior or posterior, whether at the cortex or base, also whether above or below the tentorium. The study of the pupils affords the most valuable evidence. Unfortunately the pupils are now dilated with a mydriatic for aiding ophthalmoscopic examination, but Dr. Hughes reports they were entirely normal. Normal pupils in a brain case excludes internal hydrocephalus or distention of the third ventricle. This is very important, and enables one to say at once that the region of the third ventricle is not directly involved. Hydrops of this ventricle would affect the pupils; and also the third nerve, for the pupillo-motor fibers as well as the fibers of the third nerve have their cen-

ters along the floor and sides of this ventricle.

The next question is, could these symptoms arise from hypophyseal disease? One sees so many variations from the classical symptoms of hypophyseal disease that the typical acromegaly is the rare exception. The study of this case is not complete until a transverse radiogram is taken to demonstrate the size and depth of the sella turcica, and if the clinoid processes are eroded or displaced.

Then comes the question of a tumor of the right auditory nerve. Neuroma affecting the eighth nerve would produce all of the symptoms one finds in this patient. The auditory nerve trunk is short and composed of two kinds of nerve fibers. Where these nerve fibers merge is the favorite site for the development of a neuroma. The location is usually just where the auditory nerve enters the internal auditory meatus. He ventures to predict that after Cushing reports his series of cases, their operations and ours, one will be looking for them and he will find them. The symptoms are Meniere's complex, profound tinnitus, total deafness and ocular symptoms such as choked disc, nystagmus, etc.

Cushing has diagnosed and operated on 8 cases during the year preceding the entry of America into the present war with Germany. The operation is simple. An incision is made, the scalp stripped from the temporal bone, the bone flap raised and just inside the skull lies the fusiform tumor of the auditory nerve. The dura is not opened. An incision is made in the dural sheath of the nerve and the tumor mass is curetted away. The growth is semi-benign. The amazing thing is that the hearing is restored in about 50 per cent of Cushing's cases.

The ordinary functional hearing tests are of no value. Bárány's tests must be made. The present case should be examined in this way to determine whether this disease is present. It enters into the differential diagnosis and must be excluded. The optic neu-

ritis and increased cerebral pressure are present because the tumor lies below the tentorium. Weeks' statistics published in his text book show that 100 per cent of tumors of the cerebellum have papilledema.* In almost 15 per cent of the cases the choked disc is on the side opposite to the tumor.

Next comes the question of tumor of the cerebello-pontine angle. An oncoming tumor in this location would present all the symptoms this case exhibits. Later on the hemiplegia and also the strabismus due to paralysis of the sixth nerve, as well as the palsy of the intraocular muscles from internal hydrocephalus affecting the third nerve centers, will show themselves, and then the diagnosis of cerebello-pontine angle tumor will be easy. Deafness, optic neuritis, sixth nerve palsy, third nerve palsy, hemiplegia, vomiting, headaches, etc., are characteristic symptoms of tumor in this location. The optic neuritis on the opposite side is not inharmonious with tumor of the angle in the presence of subtentorial increased pressure.

Dr. Vail said that other conditions causing monocular optic neuritis, such as tumor or abscess of the sphenoid or posterior ethmoid cavities are to be thought of and searched for, altho he hardly expects confirmatory evidence will be found favoring this group of diseases, for the other symptoms, deafness, dizziness, vertigo, vomiting and headache are not common to disease of the sphenoid and ethmoid cavities.

Dr. Alfred N. Murray said that he reported a case about 8 or 9 years ago which had symptoms somewhat similar to those in Dr. Hughes' case. The left ear was totally deaf. There was choking of the right disc. Dr. Halstead operated and found a cyst at the left cerebello-pontine angle about the size of a hen's egg. Vision almost immediately began to clear up. Vision in the left eye returned to normal, and in the right eye, where the choking was, did not return completely to normal, but remained about 20/30. There were no

typical symptoms of labyrinth disease. Total deafness in the left ear was evidently due to pressure on the auditory nerve. He saw the patient about two months ago, and his vision is just as it was when he left the hospital eight years ago. He is working at his trade the same as ever, and apparently suffers no inconvenience of any kind.

Dr. Smith said that in making a diagnosis of auditory tumor one should consider all the symptoms. There are some symptoms that are indicative of a central lesion and the patient has a tendency to fall in one direction. In locating lesions here in the temporal lobe one should bear in mind that the labyrinth apparatus and nerve fibers take a different course from those of seeing. He thinks the symptoms point directly to a right sided lesion. The beginning choked disc is absolutely indicative of some pressure. A tumor of the cerebello-pontine angle, either on the eighth nerve or in close approximation would give all these symptoms, including disturbances on the right side. There is no very marked ataxia. There is a little difficulty in coordination, but not much.

In operating he would not simply do decompression, he would go farther as one might find something that could be removed. He fails to see any difference between a cyst and a tumor. He has never been able to see any difference in making a diagnosis, from the symptoms. A spinal puncture is indicated and should be done gradually, care being taken not to let all the fluid escape at once. A cell count and Wassermann of the spinal fluid should be made. A positive colloid test would indicate syphilis. One might combine with decompression massive doses of potassium iodid, mercury or arsenic. Operation should not be delayed too long. Subtemporal decompression is the operation of choice, if one is sure there is nothing more to be found there, but the speaker would advise a cerebral decompression.

SECTION ON OPHTHALMOLOGY, COLLEGE OF PHYSICIANS OF PHILADELPHIA.

March 21, 1918.

DR. S. LEWIS ZIEGLER, Acting Chairman.

Tumor of the Orbit.

DR. WILLIAM CAMPBELL POSEY exhibited a man, aged thirty-five years, from whom he had removed a large fibroma of the orbit four weeks previously. The growth, which resembled in size and form a large myopic eye, was situated back of the eye, superior to the optic nerve, tho not in connection with it, and had proptosed the eye 12 mm. in advance of its fellow. The displacement of the eyeball was first noticed by the patient six years previously and had been slowly progressive. Vision was but little affected, tho the movements of the globe were considerably impeded. The tumor was removed thru an incision made at the outer angle of the orbit, with the intention of performing a Krönlein, if such procedure were found necessary. The delivery of the mass was effected, however, without resection of the bony wall of the orbit. Recovery had been prompt, tho there is still some exophthalmos and restriction in motion of the eyeball. Dr. Posey thought that time would gradually in large measure overcome both of these conditions.

Optic Nerve Lesions Following German Measles.

DR. POSEY read the notes of 2 cases where inflammation of the optic nerve supervened immediately after an attack of German measles. In the first case, a man aged thirty-two years, the axial fibers of the right optic nerve were alone affected, as evidenced by a slight diminution in vision, a lowering of the light sense, and the presence of a central relative scotoma. The temporal half of the optic nerve of the affected eye was grayer than normal. The left eye was unaffected. Altho there was a family history of glaucoma, there were no symptoms of this disease. Examinations of the accessory

sinuses of the nose were negative. An X-ray examination revealing an abscess of one of the teeth in the left upper jaw, the removal of the tooth was advised, but this procedure seemed to have no influence upon the neuritis.

In the second case, that of a boy, aged sixteen years, the toxins had produced an inflammation of much greater intensity, the entire head of the left optic nerve exhibiting marked papillitis, with marked involvement of the retinal vessels, the veins especially being swollen and tortuous, giving rise to the suspicion of thrombosis. As has been observed by others in similar cases, vision was but little affected, $V. = 5/7\frac{1}{2}$. The visual field was normal, save for a relative central scotoma, probably accounted for by a few fine hemorrhages in the macular region.

Dr. Griscom spoke of a case of intense double optic neuritis following measles which he had reported in 1911. The patient made a complete recovery both as to vision and fields, altho the nerve heads remained very pale. He reviewed the literature on the subject up to the time of the report and found only 23 cases of blindness due to optic nerve lesions following measles, all but three of which came under the head of optic neuritis. These cases could be divided into three classes: (1) Those showing evidences of primary cerebral involvement with secondary optic nerve change; (2) those showing meningitis as the most prominent symptom with consequent optic neuritis, and (3) those showing optic neuritis without any other local or general symptoms.

Associated Action Between a Paretic External Rectus of the Left Eye and the Masseter Muscle.

DR. POSEY exhibited for his colleague, DR. SCHWENK, a man, aged thirty-four years, who showed a curious contraction of the paretic external rectus muscle of the left eye, when the masseters were thrown into activity by the act of biting. (See full report, p. 393.)

Occlusion of Central Artery of Retina.

DR. F. KRAUSS presented a case of occlusion of the central artery of the retina of the right eye in a fifty-eight-year-old man. A small section of the retinal artery was discernible on the disc, otherwise there was no evidence of its existence. The veins were full and tortuous, with innumerable hemorrhages thruout the field. The vision six weeks after the onset was 3/60, which is remarkable considering the fundus change.

Angioid Streaks in the Retina.

DR. CHARLES R. HEED exhibited C. J., male negro, aged twenty-eight years.

Ophthalmoscopic examination: R. E., media clear, optic disc and retinal vessels normal. The temporal half of fundus, extending from the disc margins in a radial direction for a distance of four diameters, presents retinohoroidal changes of an unusual type. The color is grayish, presents a few pigmented areas and numerous hemorrhages lying in the deeper retinal layers. Many deeply pigmented branching streaks running beneath the retinal vessels are noted. These streaks appear to come into direct contact with many of the hemorrhages and converge to the pigment area about the disc. There are a few hemorrhages seen in the nasal field with several of the streaks converging to the disc margin. The general nasal and entire peripheral portions of the fundus appear normal. A dense capsular opacity obscures the fundus of the left eye.

Apparent Accommodation in Aphakia.

DR. WILLIAM ZENTMAYER reported a case in a girl, aged eight years, with acquired cataract. This report is published in full, p. 570.

DISCUSSION.—DR. Ziegler said that the cases he had observed of this condition were due either to the action of the extraocular muscles on the globe or to nipping of the lids, and also that such cases were those with a correction of +13 D or more, with a very small amount of astigmatism.

J. MILTON GRISCOM, M. D.,
Clerk.

COLORADO OPHTHALMOLOGICAL SOCIETY.

April 20th, 1918.

DR. D. H. COOVER, Presiding.
Retinal Angiosclerosis.

Dr. D. A. Strickler presented Mrs. L. D. W., age 57, housewife. First seen March 30, 1918, with the following history: Two or three months ago she suddenly noticed dimness of vision in R., but no pain in the eye or head; thinks she sees a little better than when first affected, but no appreciable change recently.

Family history: She has two brothers and three sisters ranging in age from 53 to 63, all living. Mother died of Bright's disease at 59. Father died at 74 of paralysis. Had had three or four attacks in the two years before death.

Examination: V = L. 20/100, corrected to 20/20. R. central vision lost with indefinite vision peripherally. Right fundus showed small retinal hemorrhages between disc and macula, also on the upper portion of the disc, with a few white lines especially toward the macula. No definite lesion was noted in macular region. There was undue tortuosity, alteration in size, breadth and course of retinal arteries; loss of transparency, as shown by veins, lost to view under the crossing of artery; and, impeded venous circulation at the distal end. In the left fundus there were small, hemorrhagic spots above disc about 1 and $\frac{1}{2}$ diameters, with other conditions much the same as in R.

Diagnosis: Retinal angiosclerosis with retinal hemorrhages. Blood pressure 185-220. A trace of albumin, with casts. Hypertrophied heart.

Examined next on April 18th when she reported that on April 3rd, R. E. became red, painful and sensitive to light and had so remained. Tension definitely increased; the fundus could not be seen because of cloudy media. Diagnosis: Hemorrhage into vitreous.

DISCUSSION.—Dr. J. A. Patterson said he had had a similar case only he believed his patient's eyes were worse.

This patient, a man, had suffered from smallpox. While in quarantine his eye gave him trouble and he used some atropin, which was given him by another patient, also in quarantine. The atropin was used for one week. After he was out of quarantine, Dr. Patterson found the T. 68 mm. of mercury. Eserin was immediately used. The eye and vision were better for a time, but later were worse again. Under hyoscin and dionin the eye was made to improve a second time. On account of the hyphema, he believed an operation was not indicated. There were posterior synechiae, and the fundus could not be examined on account of the cloudy media, hemorrhage, etc.

Dr. E. M. Marbourg spoke of Dr. Patterson's case, as he had the pleasure of seeing it, too. At the time he made his examination, the tension was 64 mm. and the pupil was 4 mm. in diameter. The patient had lost weight. Under dionin and eserine, the tension was reduced to 45 mm. but the pain recurred. At no time was he able to obtain a view of the fundus.

Dr. Melville Black said the hemorrhage of the optic nerve or pressure on the central fibers from the sclerotic central artery may have produced the loss of central vision in Dr. Strickler's case.

Serpent Ulcer of Cornea Due to Pneumococci.

Dr. Wm. C. Bane presented A. J. G., age 33, woodworker. On April 13th, he got a bit of sawdust in O. S. When first examined April 15th a small ulcer existed. This was curetted; antiseptic ointment applied; and the eye sealed. The next day the ulcer was 2 mm. in diameter, and the eye had been very painful during the night. Prince's pasteurization method was used and atropin instilled. Aspirin and calomel were given. April 17th the ulcer was 3 mm. in diameter; there was a crescent of exudate in the anterior chamber and marked congestion of the eyeball. So the pasteurization was repeated. At 4 P. M. the ulcer was larger and the discharge revealed

pneumococci. Under cocain, ethylhydrocuprein hydrochlorid powder was applied direct to the ulcer. Marked reaction followed, the epithelium around the ulcer becoming detached. April 18th, had a comfortable night with no increase of exudate in the anterior chamber. Ulcer 4 mm. in diameter and a ring $1\frac{1}{2}$ mm., devoid of epithelium, around the ulcer. Atropin and iodoform were used. April 19th less exudate in the anterior chamber and less congestion of the ball. Epithelium about reformed around the ulcer. April 20th ulcer nearly 3 mm. in diameter and no exudate in the anterior chamber. One per cent ethylhydrocuprein hydrochlorid instilled. Atropin and iodoform used. Eyeball not painful.

DISCUSSION.—Dr. E. R. Neeper said there were two interesting features in this case. First, he had not known that optochin could be used in powdered form, as he felt it was too escharotic when so used. Second, cocain had never been very satisfactory in his hands in any case, as it tends to make the infection spread and it makes the cornea quite hazy. He believed it lessened the vitality of the corneal epithelium if not other tissues of the cornea. He preferred to use holocain in such cases.

Dr. Melville Black said optochin was supposed to produce amaurosis in pure form. At least this had been true in the case where large doses were used in the treatment of pneumonia. The overhanging edges of the ulcer were gone in Bane's case, probably due to the fact that he had used the powdered optochin. He believed this feature alone made it worth while to use the powdered form.

Dr. Wm. H. Crisp also emphasized the fact that amaurosis occurred only with systemic poisoning where large doses of the optochin were used in pneumonia. It is a copper salt and hence is apt to produce small vesicles of the cornea. He doubts if the powdered optochin were better than pure nitric acid if carefully used.

Dr. O. Orendorf pointed out the fact that young adults recover from pneumococcus ulcers much quicker than the aged.

Dr. J. A. Patterson said he rarely bandaged an eye. He believed if we seal an eye we prevent drainage and favor a pneumococcus infection. He used Dr. Crisp's method of rubbing in the optochin.

Dr. Wm. C. Bane said he favored the use of melted vaselin poured into the conjunctival sac. He melted this by warming it in a teaspoon, over an alcohol flame, until it was liquefied, and then poured it into the conjunctival sac. He stated that he did not use White's ointment, but simply plain sterile vaselin.

Partial Cataract Following Injury.

Dr. Edward Jackson presented a man, aged 68, a stone cutter, whose left eye had been struck with a minute piece of stone twenty years ago. He was in central Wyoming at the time and was not seen by any doctor. The eye was "blind for three weeks" and then gradually got better. Before the accident, it had been as good as the right, but since then it had been noticeably worse. Three months prior to the meeting, vision in R. became impaired, so that L. was the better eye. Corrected vision: R. = 0.4; L. = 0.7. The latter required $+1.50 \text{ C} - 3.00 \text{ cyl. ax. } 65$. O. D., presented retinal vascular disease, dilated veins, numerous small hemorrhages, retina and optic nerve gray and opaque with exudate. Urine normal; blood pressure, 160—110. The keratometer showed $+0.50 \text{ cyl. ax. } 180$.

The left eye showed slight narrowing of the arteries and narrowing and obscuration of the veins at crossings. It was free from inflammation, iris and pupil normal, cornea clear except a faint nebula in front of nasal margin of a 5 mm. pupil. In the lens there was a comma shaped opacity. The head deep in the anterior cortex or in the anterior layers of the nucleus, looked white like a calcareous deposit, with a small black speck. The tail of the opacity extending forward, and toward the

center of the pupil was thinner and gray. In the lower nasal periphery of the lens, there were masses of cortical opacities anterior; but these did not extend into the pupil. The remainder of the lens was clear. The right lens was clear. The probable explanation seemed to be a minute foreign body lodged in the anterior nucleus of the lens, followed by some swelling and opacity, clearing up subsequently, and the eye becoming entirely quiet, and remaining so for twenty years.

DISCUSSION.—Dr. J. A. Patterson asked if the lens had retained a foreign body. Dr. Jackson thought this was possible if the foreign body had been very small and nonirritative.

Dr. Melville Black said twenty years' duration went to show how long a lens will retain a foreign body without complete cataractous change. He referred to a case of a child struck in the eye with a piece of copper following the explosion of a miner's cap. The iris and lens were both damaged and there was a partial opacity of one lens. In another case the slightest damage may produce a total opacity, so we cannot always judge by the extent of the injury how much obscuration of vision will result.

Tests for Color Blindness.

Dr. Edward Jackson exhibited a set of colored plates devised by Ishihara of Tokyo, Japan, for testing color vision. Each plate consisted of a background of a confusion color, with a figure in the test color easily recognized by the normal sighted, but unseen by the color blind. In two tests, the colors were so chosen that the figure would be more evident to the color blind than to the normal sighted; and one plate was arranged to be equally visible to normal or color blind.

Interstitial Keratitis.

Dr. W. F. Matson presented Miss R. O., age 32. Father and mother were living and in good health. Several brothers and sisters were living and have always been well. One child died in infancy with some kind of bowel trouble. Patient had had the best of health, she thought. First attack came

July, 1917, and continued over a period of 10 weeks, then cleared up with 20/20 vision with her correction. This was in the upper segment and some small vessels still remain. The last attack began three weeks ago from the nasal sector and had extended $3/4$ across the cornea. The upper sector, which became involved in the last attack, was slowly getting clearer.

The upper third molar was devitalized seven years ago, and this may have a root abscess as the cause of the interstitial keratitis. The tonsils and sinuses were negative. Tuberculin and Wassermann tests had not been made.

DISCUSSION.—Dr. J. A. Patterson said the dark color of the tooth was due to the devitalization and not to the amalgam filling.

Dr. G. F. Libby said Dr. R. C. Brownlie believed a dead tooth was a menace to any patient's health and should be removed to avoid a future streptococcus infection.

Dr. Melville Black said this patient had a uveitis due to any one or more of four causes. First, infection in the tonsils; second, gastrointestinal infection; third, tuberculosis; and fourth, root abscesses about the teeth. He believed that all four of these were very important and should be thoroly investigated. He would first have her teeth X-rayed and if any were at all suspicious they should be removed. Then he would have a complement fixation test made. If positive for tuberculosis he would begin with very small doses of tuberculin, first human; second, bovine, but he would be careful to avoid a focal reaction.

Penetrating Eye Injury.

Dr. Wm. C. C. Bane presented Mr. E. C. C., aged 33, blacksmith. On March 22, 1918, while striking iron with a hammer something struck left eye. There was not much pain at the time, but considerable bleeding. The eye was examined two hours after the injury. Could see hand at 18 inches. There was an oblique cut 6 mm. in length through the limbus at the superior nasal quadrant with prolapse of the iris and hyphema. The prolapsed

iris was excised. The examination with the sideroscope was negative. The X-ray located a foreign body 10 mm. above the horizontal plane of the cornea; $\frac{3}{4}$ mm. to the temporal side of the vertical plane of the cornea; and, 32 mm. back of the center of the cornea in the orbital tissues. The foreign body had passed thru the eyeball. It measured 2x4 mm. on the plate.

There was freedom from pain and some improvement in vision until the 6th day when the eye became congested and painful. On the 7th day the eye was very painful and on the morn-

ing of the 8th day the pain continued. The eye was congested and there was an exudate showing in the lower part of the anterior chamber with some photophobia of the fellow eye. The enucleation was performed during the afternoon of the 8th day. Examination of the eyeball revealed a posterior scleral wound where the metal had passed thru. The recovery was rapid, and the patient was wearing an artificial eye at the end of the second week.

FRANK R. SPENCER, M. D.

Secretary.

PHYSIOLOGIC OCULAR TENSION.

(La tension oculaire physiologique.)

DR. A. MAGITOT.

PARIS.

A review of the mechanism controlling the ocular tension in normal eyes, and its reaction to drugs and experimental interference which emphasizes the vascular factor in regulating the intraocular pressure. Abstract-translation from *Annales d'Oculistique*, v. 154, pp. 272, 334, 385, by M. W. Fredrick, M. D.

The subject of ocular tension is a part of normal physiology; it is worth while insisting on this, inasmuch as most treatises on the subject run over into glaucoma and its therapeutics. The fact that the explanation of certain pathologic conditions is still wanting is due to the very narrow view taken of the aqueous and the ocular tension.

It is a matter of common belief that the intraocular pressure is greater than the atmospheric pressure. If a freshly enucleated eye is placed with the cornea upwards and an opening made into the cornea, the endocular fluid will escape. The eye being that organ which receives luminous impressions it must have a constant shape, and must be sufficiently rigid to resist the traction made on it by the external eye muscles. Altho the sustaining coat is dense the eye retains its form only so long as the endocular liquids are not lost by diffusion or evaporation.

THE METHODS OF MEASURING INTRAOCULAR TENSION ARE TWOFOLD.

(1) The direct, or laboratory method. This method is not practical in living human subjects, as it requires the introduction into the eye of a hollow needle, and the production of an immobility not obtainable by ordinary anesthesia on account of the action of the anesthetics on the heart. One would be obliged to resort to curarisation and artificial respiration with all the cumbersome machinery used for this purpose in the physiologic laboratories. The manometric methods follow either Mariotte's law of the compressibility of vapors, or they work with a tube open to the air. The best measuring device for this purpose is Wessely's improvement on Schulten's device, in which a small capsule is interpolated between the manometer and the eye and marks the findings on a blackened disk.

Ever since Brissaud, in 1709, made his first determinations of hypertension by digital palpation, clinicians have sought better means for determining the intraocular tension. Even the method of Bowman, which is still used too much by ophthalmologists who have learned to use their fingers and have never resorted to instruments of precision, is but a makeshift, as we are dealing with the personal equation, and even strong variations may escape detection. It was but logical that some instrument should be sought which would replace the fingers of the clinician, and this lead to

(2) The indirect method and the invention of the tonometer. In 1863 v. Graefe, Haemmer, and Donders, and in 1865 Dor, each invented an apparatus for this purpose. The many others appearing later proved that none was satisfactory until the well known tonometer of Schiötz made its appearance. The principle employed in tonometers is either that of measuring the force necessary to cause a certain depression in the sclera (Donders, Fick, Nicati), or of measuring the depression in the cornea caused by the application of a fixed pressure (Imbert, Mallakaff). In the Schiötz tonometer both principles are used; it is constructed on the principle of the sphero-cylindrometer. Even this instrument has its limitations, as it is an index of the interior pressure and the depressibility of the ocular coats; and its reading would be completely exact only if:

(1) The elasticity of the ocular coats were the same in the living and the cadaver (it changes very quickly after death);

(2) If the elasticity were the same in all individuals;

(3) If this elasticity did not influence the oscillations of the apparatus. Too much importance, therefore, must not be accorded the readings.

NORMAL OCULAR TENSION AND ITS VARIATIONS.

Wahlfors, using a manometer, found a tension of 25 mm. Hg. in a human eye. Toczynski, in a recent review of the results found by his predecessors, obtained the following averages:

In normal subjects between 1 and 25 years, 18.66 mm. Hg.

In normal subjects between 26 and 50 years, 17.48 mm. Hg.

In normal subjects between 51 and 75 years, 17.17 mm. Hg.

It is a well established fact that ocular tension varies with the different animal species: thus it varies in the guinea pig between 10 and 15 mm., in the rabbit between 18 and 25, in the cat between 22 and 30, in the dog between 15 and 25, and in man between 12 and 30. It is worthy of note how small a role the bodyweight plays in the ocular tension. Thus, while there is a difference between the tension in the guinea pig and man, it is but small compared with the difference in body weight, and the cat seems to have about the same ophthalmotonus as man. In this regard it is of interest to note whether there is a relation between blood pressure and body height; thus we find:

Guinea pig (carotid) 111 mm. Hg.

Rabbit (carotid) 100-140 mm. Hg.

Cat (carotid) 130-150 mm. Hg.

Dog (carotid) 140-180 mm. Hg.

Man (femoral) 110-120 mm. Hg.

(In the human aorta the pressure is estimated at 150-200 mm. Hg.)

Blood pressure varies according to racial and biologic differences. So does the ocular tension. While the mean tension in man may be taken as 20 mm., in the northern peoples, according to Stock and Langehan, it is higher; and may vary between 12 and 26 mm. Sex makes no difference. Altitude, however, has a marked influence. Uribe Troncoso has recorded a difference of several mm. between rabbits living on the Mexican plateau and those living in the lowlands. Nicati has shown that in diving bells, under a pressure of two and one-half atmospheres, the ocular pressure was almost doubled in rabbits, and was increased one-fifth in man.

A constant figure is impossible both in man and animals, as the pressure varies not alone with race and age (Wegener found 30 mm. frequently in children as against the average of 15 mm. in old people), but may vary in the same

individual from day to day. Ruata found a difference of 5 mm. between the morning and evening readings in the same person, with parallel variations in the blood pressure of 5 to 10 mm. As with the blood pressure the intraocular pressure is lowered by fasting and anemia; and increased by exertion, inspiration, and asphyxia. Contraction of the extrinsic eye muscles increases the tension, the muscles innervated by the third pair being more influential than those innervated by the fourth pair, according to Wessely, Lederer and Levinsohn, and contrary to Groenholm's findings. Levinsohn finds that the contraction of the intrinsic eye muscles raises the tension because it increases the circulation, but Hess and Heine could not confirm his results in their observations.

ANATOMIC DETAILS.

The outer wall of the eyeball, the cornea and sclera, are not absolutely rigid, but are elastic within very narrow limits. There is no rigid tissue in the body except bone, and all connective tissue is more or less extensible, especially when it contains elastic fibres. Even in those animals in which there are large inserts of cartilage and bone in the sclera the cornea still makes changes in form possible. This elasticity of the outer tunic is, as already stated, of low degree, as shown by the experiment of Koster, who found that to raise the tension in a rabbit's eye from

5 to 10 mm. Hg. meant the introduction of 6 mm. of liquid.

10 to 20 mm. Hg. meant the introduction of $3\frac{1}{2}$ mm. of liquid.

20 to 30 mm. Hg. meant the introduction of 1 mm. of liquid.

30 to 40 mm. Hg. meant the introduction of $\frac{3}{4}$ mm. of liquid.

40 to 100 mm. Hg. meant the introduction of $\frac{1}{3}$ mm. of liquid.

The choroidal tunic is much thicker in the living than in microscopic sections owing to the collapse of the choroidal vessels. This thickness is not 50 to 80 μ , as determined by R. Greeff in man, but 200 to 300 μ (Venneman), or even as high as 300 to 350 μ in frozen sections taken from the neighborhood of the optic disc in the new-born (Wolfrum). In

old people, due to the sclerosis of the vessels, the thickness of the choroid is reduced by one-half. This great vascular development of the choroid is not intended to maintain the temperature of the retina, so as to insure the proper functioning of the retinal nerve elements, as this is amply provided for by the surrounding blood vessels and tissues, such as the muscles, orbital fat, eyelids, etc. The real purpose of this large arterial supply and of the venae vorticosae, for which there is nothing analogous in the pia mater of the cerebral centers, is the maintenance of a tonus sufficient to counterbalance the action of the extrinsic muscles, or of any predominant set of muscles, and thus to assure to the eye its form. The choroid must, therefore, be considered an erectile organ, identical with the *rete admirabile* of the fishes, the so-called choroidal gland. (The significance of this choroidal gland is not clear. It has been supposed to have to do with the accommodation, but it is more likely to be a regulator of the pressure of the eyelids according to depth. Amphibia have a much thicker eyecup than terrestrial animals). The choroid contains a considerable number of elastic fibers in the lamina fusca, the walls of the larger vessels, and in the chorio-capillaris. Iwanoff has found non-striated muscle fibers accompanying the vessels. Besides, the cavernous system of the choroid has a nerve regulating organ, whose branches, both medullated and non-medullated, form a network in the lamina fusca, and receive numerous branches from the ciliary nerves, the whole constituting the vasomotor apparatus for the arteries and the chorio-capillaris. The choroid has, furthermore, a number of ganglion cells of the sympathetic type (multipolar). This assembly of nerve cells constitutes a veritable intraocular ganglion (H. Muller), the regulatory importance of which will be shown later on.

We must keep in mind that we have two independent territories in the vascular system of the eye: one caring for the retrobulbar segment and the retina; and the other for the uveal tract and the sclera, including some of

the episclera. The most important source for the uveal tract are the posterior ciliary arteries, about twenty in number. The posterior long and the anterior ciliary arteries contribute in a modest way to the arterial supply. The veins are two or three times more numerous than the arteries, and are of much larger caliber. They arise suddenly from the fusion of about fifteen capillaries which meet in a small vortex, and in turn form larger vortices, called *venae vorticosae*. The anterior segment of the eye is also rich in veins, for instance the ciliary region, the ciliary processes being almost entirely made up of bunches of veins. In spite of the seemingly large number of anastomoses between the anterior and posterior venous systems, the suppression of either leads to a marked venous stasis of the eye.

RELATION OF THE OCULAR TENSION TO THE GENERAL CIRCULATION.

While the tension of the eye will, generally speaking, be lowered by a diminished flow of blood from the internal carotid, and increased by an impediment to the venous flow, we must not forget that the eye has a local regulating system, which allows it to equalize and distribute the blood within its walls according to local needs. This contractile reservoir will be called upon to equalize variations of pressure coming from outside the eye, so that the eye is under the influence of the general blood pressure up to a certain point only. This explains how the eye can maintain a tonus necessary for its efficient functioning, and why a rise of 80 mm. in the carotid pressure will cause a rise of 8 to 10 mm. only in the ophthalmotonus. Another thing to consider is that the blood pressure sinks as we approach the periphery, so that while the blood pressure in a dog's carotid is 140 mm. it is 130 in the *dorsalis pedis*, and much less in the ciliary arteries.

While the pressure in the jugular is negative, a fact which makes us fear the opening of the larger veins of this system, the emissary veins of the eye are under positive pressure, possibly

of 8 to 9 mm. Hg. But the pressure in the veins within the eye must be much higher, otherwise they would flatten under the intraocular pressure of 20 mm., thus showing the importance of the regulating reservoir within the choroid. Apart from these local phenomena we may assume that the ocular tension stands under the influence of the general circulation, and responds to the anemia and stasis which manifest themselves in the large vessels.

Slowing of the heart or opening the thorax bring about hypotension. Stopping of the heart in a rabbit lowered the tension from the normal tension of 18 to 25 mm. to 8 mm., and Nicati has seen a like result produced in man by syncope or death, as measured with his sclerometer. Section of the spinal marrow, which suppresses the principal excitator of the heart, or the stimulation of the depressor nerve of Cyon brings about the same condition. In a rabbit, in which the ocular tension had been raised to 64 mm. by artificial inflammation, cutting the vagus and exciting the stump caused a drop of the ocular tension to 34 mm., the blood pressure dropping from 140 to 80 mm., the two curves showing an absolute parallelism and synchronicity.

The percentage of drop in the ocular tension will, however, be greater if the ocular tension was above normal before the experiment, and will then more closely approximate the percentage of drop in the blood pressure. If a difference in tension existed between the two eyes the proportional drop will be much greater in the eye showing the higher tension. Tying one carotid in a rabbit with an initial tension of 20 mm. produced a drop of 5 mm.; tying both carotids produced a drop of 6 mm. To explain the small amount of decrease we must remember that in the rabbit the blood supply for the eye comes largely from the vertebral arteries, so that the drop is not as great as it would be were it not for this accessory supply. The drop is temporary, and, if the time of compression does not exceed one minute the tension rapidly resumes its former height.

As a corollary to the decrease of tension due to decreased blood supply one would think that an increase in the blood supply would give rise to an elevation of ocular tension; this it does, but in a far less degree than the decrease in blood pressure, thus again proving the influence of the regulating organ situated in the eye. Aside from the elevation due to rapid inspiration and exertion, changes which can hardly be noted with precision, Heine has shown that injecting artificial sera into the veins of a rabbit sends the blood pressure up to 300 mm., and the intra-ocular tension to 100 mm. Hg. In a rabbit, in whose left eye the tension had been raised from 20 to 43 mm. by subconjunctival injection of a salt solution, compression of the abdominal aorta raised the tension in the untouched right eye to 26 mm., and in the left eye to 47 mm., showing that the relative rise in tension is greater the lower the tension was before the augmentation of blood pressure. Were we to accept the classic theory that the hypertension is brought about by an active secretion of the aqueous humor, we would be forced to assume that an increase in the liquid contents of the eyes would exaggerate an already high tension; we find, however, that an eye already under high tension seeks to defend itself against a still higher tension.

Section of the heart moderator nerve in a curarised cat (Wessely), raised the blood pressure from 150 to 180 mm., and the ocular tension from 25 to 28 mm. Exciting the cut end brought the pressure and tension down, but they regained the former figures when the excitation ceased. Thus, altho there is a parallelism between the two curves, the increase in ocular tension is small. Compression of the superficial cervical veins has little effect, as the return flow thru the medullary veins is ample. In order to have any marked effect on the ocular tonus one would have to compress all the cervical veins, which is difficult to do, an approach to which is made in the Bier method, in connection with which Renner, Hoppe, and Schirmer studied the

effect in man of cervical compression, and found it without influence on the ocular tension. Whereas Wessely agrees with Schultze, after a series of experiments on curarised animals, in whom he determined at the same time the blood pressure in the carotid, as variations in the blood pressure would invalidate the eye findings. He found that cephalic venous stasis causes a slight rise in the ophthalmotonus, which he attributes to exophthalmus.

While, in a general way, the blood pressure and the ocular tension show a certain amount of parallelism, the disassociation of the two is not alone possible, but frequent. The low tonus in old people has already been mentioned. It is surprising to find normal, or even lowered, tonus in nephritics, subjects of hypertension, and cardiorenals. While the injection of serum into the veins produces a marked rise in both blood pressure and ophthalmotonus, the injection of adrenalin into the veins while increasing the blood pressure in the larger vessels considerably, produces either no change in the ophthalmotonus or causes it to fall, as a result of the vasoconstriction which takes place in the ocular vessels, thus again evidencing the importance of the physiologic individuality of the eye.

(5) RELATION OF OCULAR TENSION TO THE LOCAL CIRCULATION.

As the manometer cannot be applied to the small vessels constituting the erectile tissue of the uvea, we are obliged to draw conclusions from other well known facts. We know that an intraocular tension higher than the vascular pressure would suppress the local circulation. Pressure on the globe increases the tonus of the globe and brings into play venous pulsation which is not perceptible under normal conditions. If we suppress the blood circulation in the eye there results an immediate lowering of the ocular tension, as, for example, we have in death. In a freshly killed rabbit the tension sank from 20 to 8 mm. Hg. By injecting Ringer's solution into the carotid, O. Weiss restored the tension of 20 mm. Hg.

T. Henderson thinks the intraocular pressure is quite comparable to the intracranial pressure as studied by Hill, and shown by him to be equal to the venous pressure; and recalls the fact that liquids always tend to adjust themselves to the lowest level. The comparison of the bony skull with the eyeball is unfortunate, as the sclera possesses a certain amount of elasticity, even though that may vary. O. Weiss' reasoning is more alluring: If in a dog's aorta, the blood pressure is 170 mm. Hg., dropping to 140 in the carotid, and to 130 in the dorsalis pedis, it should be 50 to 70 mm. Hg. in the ciliary arteries during the diastole, and suffer a further loss to 40 to 60 mm. Hg. by capillary reduction. When we remember that the normal intraocular pressure in the dog is 15 to 25 mm. we see that the difference between the two pressures is sufficient to preclude compression of the ocular vessels. While we have no means of verifying Weiss' figures we must assume that they are very near the truth; altho they may be too high, as we know that a very moderate compression of the eye will make the venous pulse appear.

(A) THE OCULAR PULSATIONS.

Although neither felt by palpation nor seen with the ophthalmoscope the oscillations due to the impulses of the choroidal blood flow can be perceived by the tonometer of Schiøetz, and still better by the registering manometer. On a rabbit Wessely found the amplitude of oscillation 0.4 mm. with an ocular tension of 25 mm. Hg.; at 45 mm. the amplitude for 1.4 mm.; and, with a tension of 70 mm. Hg. it was 2.5 mm. When the ocular tension sank below 15 mm. no oscillations were registered. This explains the fact that the tonometer needle does not always show oscillations. The graphic method shows the ocular pulse to be slightly behind the carotid pulse.

If in a curarised rabbit, in whom the carotids show a fixed pressure, ocular hypertension is induced by hyperemia, the higher the ocular tension goes, the wider the oscillations of the ocular pulse. The increased amplitude of os-

cillations cannot, therefore, be attributed to the beating of the orbital arteries, we must assume that it is the greater or lesser fullness of the eye vessels which give rise to (a) ocular hypertension; (b) ocular pulsation. Without this, one could not understand how an intraocular tension of 70 mm. Hg. could exist without a complete flattening of the choroidal vessels.

We must not confuse these "active" ocular pulsations with those which we see with the ophthalmoscope when progressive digital pressure is applied to the eye. We then see pulsations first in the veins and then in the arteries; finally the arteries become empty. These pulsations are always present, and may be made perceptible by using the apparatus of Gullstrand (Speyr); pressure simply increases their amplitude. We are here dealing with "passive" pulsations in nonencapsulated vessels which pass thru a rigid strait, the lamina cribrosa. Attention is called to the fact that the beats are manifest in the most central part of the papilla and reach the borders of the papilla in exceptional cases only. The mechanism of this can be understood if we remember that the normal jugular pulse is due to the period of pause in the blood wave at the moment of the auricular systole.

(B) ACTION OF THE ARTERIAL PLEXUS.

It is quite evident that the retinal territory is without influence, and that the suppression of the central artery and its network has nothing to do with tension. The changes which we see in these vessels before or after a glaucomatous crisis are simply coincident with a generalized angiosclerosis. Entirely different is the import of the ciliary network, which Wagenmann was about the first to subject to experimental section and ligature. Unfortunately Wagenmann's experiments were made on rabbits whose anatomic formula is slightly different from that of man, inasmuch as the rabbit has no anterior ciliary arteries and its posterior ciliary arteries are terminal.

Wagenmann found that section of a long and short posterior ciliary artery

induces only transitory hypotonus, because the other arteries undergo a compensatory hypertrophy. If, however, all the long and short posterior ciliary arteries are tied, the globe at once becomes flabby and soft, the anterior chamber disappears, and the eye atrophies. The choroidal plexus has been deprived of its entire blood supply. In man the operation of opticociliary resection, recommended for certain forms of glaucoma, has afforded us an opportunity of studying the effect of sectioning the posterior ciliary arteries. But the results are of small value, inasmuch as the hook is blind, and the operator cannot feel just which vessels he has cut; furthermore, the accompanying section of the nerves often gives rise to a keratitis. This much is certain, that suppression of an important blood supply diminishes the tension of the globe.

(C) ACTION OF THE VENOUS PLEXUS.

As already stated there are two venous territories in the eye; the posterior made up of the *Venae vorticosae*, and the anterior made up by the anterior ciliary veins. Contrary to the classical opinion that the posterior territory is the only one of importance to the eye, Henderson has shown that the anterior group also plays a considerable role in the physiology of the eye. Adamuck seems to have been the first to occupy himself with the vorticosae veins. After ligating these vessels he noted a rise in tension to 70 mm. Hg. in the rabbit; and to 90 mm. Hg. in the cat. Koster and Weber obtained like figures; but they found that it was necessary to ligate all four vorticosae veins to obtain a lasting result. Otherwise the hypertonus changes rapidly into a hypotonus, followed by pigmentary lesions of hematogenous origin, modifications of the pigment epithelium, alterations in the visual cells, and obliteration of the anterior chamber with iridic adhesions. Arlt, Exner, and Van Geuns came to similar conclusions.

IMPORTANCE OF THE ANTERIOR CILIARY PLEXUS.

In 1905 Bartels, while practising the subconjunctival ligature of the tendin-

ous insertions of the recti muscles in dogs, noted that after the ligation of the last tendon the tension rose to 80 mm. Hg. and more. Several hours later marked conjunctival edema set in and the retinal vessels showed narrowing. This was followed by hematomata near the limbus, the cornea became cloudy and anesthetic. There was dilatation of the pupil, but the anterior chamber suffered no change. Thus we see the ligature of the anterior ciliary veins produces a greater hypertension than the suppression of the vorticosae veins; and one which is more lasting. It was watched during five months in a dog, without producing papillary excavation or obliteration of the iridocorneal angle. This proves the importance of the return thru the anterior veins, and the small value of the anterior ciliary arteries. These latter, as already stated, are absent in the rabbit, but luxating the eyeball will produce hypertension in the rabbit because of the constriction of the tendinous insertions of the muscles by the eyelids.

(D) ACTION OF ENDOCULAR HYPEREMIA.

The methods of inducing hyperemia of the globe are twofold: those inducing passive hyperemia, and those inducing active hyperemia. The hyperemia due to the ligation of the anterior and posterior venous territories has just been described. Another method, and one which possesses considerable clinical interest is the method of inducing hyperemia by suction. Wessely, using a curarized animal, showed that the pressure in the carotid remained fixed, but that the ocular tension rose sharply, attaining its height in 30 to 40 seconds, whence it slowly receded. If the suction is discontinued there is a sudden decrease in tension, which sinks to a lower level than that which obtained before beginning the experiment.

Often a very intense exophthalmia is produced, accompanied by chemosis and edema of the lids. This is due to the stasis in the periocular tissues, which is sometimes so considerable that orbital and palpebral hematomata

appear. This stasis may be responsible to a certain degree for the rise in eye tension, by compressing the emissary veins of the eyes. The main reason for the rise in tension is the extreme repletion of the erectile tissue in the uvea; which is so great that the vessel walls are taxed to their utmost. The sudden onset of the hypotension when suction is discontinued led to the hope that this might be an important therapeutic agent in the treatment of glaucoma; but here again the difference in the conduct of a healthy and a diseased organ was overlooked. The hypotonus, due to an acute distention of the globe, is but temporary, and is gradually replaced by a tension which is even higher than the initial tension.

Active hyperemia can be brought about by the mechanism of inflammation. There are many agents for accomplishing this, the least painful being sodium chlorid, which has been largely used clinically. It is of interest to the physiologist, inasmuch as it provokes a reflex vasodilatation. A 0.7 per cent solution is absolutely without effect, hence the inefficiency of normal physiologic serum. To produce an effect on the ocular tension one must use solutions of at least 5 per cent. Above this strength the more concentrated the solution the more violent and lasting the effect. Wessely has shown that one cc. of a 5 per cent solution produces the same effect as $\frac{1}{2}$ cc. of a 10 per cent solution; but that a cc. of a 10 per cent solution produces a much greater hypertension.

The explanation for this lies in the fact that the organism does its own diluting, so that in three-quarters of an hour a 5 per cent solution has been diluted to a 0.7 per cent solution, that is, an isotonic solution. Starting with 20 mm. Hg. in a curarized rabbit the injection of $\frac{1}{2}$ cc. of a 10 per cent solution of salt started an increase in tension after one minute; and in fifteen minutes a tension of 60 mm. had been reached. After remaining stationary for a while the tension sank to a point below the initial tension; but this last phenomenon may be due to the general loss of pressure in an animal after pro-

longed curarization, as it was found in the other eye also.

That this hypertension is not due to osmosis into the anterior chamber of the sodium chlorid is proven by Wessely, who found the sodium content of the anterior chamber unchanged in living animals, as long as the strength of the solutions did not exceed 15 per cent. Whereas, in the dead animal, an injection of a 5 per cent solution considerably increased the NaCl. content of the anterior chamber, caused hypotonus, and opacification of the cornea. That the hypertonus is not due to venous compression brought about by the distention of the tissues is proved by the fact that a 0.7 per cent solution is inoperative. That it is due to internal hyperemia is proved by the fact that an intravenous injection of adrenalin will arrest a beginning rise of tension. The effect of injecting under the conjunctiva various chemical substances will be regulated by their ability to set up a reflex vasodilatation in the interior of the eye. Witness the injection of sublimate.

But, it is not necessary to use these liquids in subconjunctival injection to get this result. Instillation of irritating liquids into the conjunctival sac will suffice. So will traumata of the cornea, or cauterization of the cornea or limbus. All these procedures increase the albumin content, and bring into play the antibodies. This appearance in the aqueous of substances which are normally found in the serum must be interpreted as the transudation of serum thru the dilated capillary walls. The increase in tension and the passage of albumin into the aqueous are parallel phenomena, both due to intraocular hyperemia.

(6) INNERVATION AND OCULAR TENSION.

This is a very intricate chapter. The innervation of the eye is one of the most complex subjects of anatomo-physiology, on which but little light has been shed by the studies published so far. The cause for this lies in the fact that experimental research on the human being is impossible, and that the studies on "laboratory" animals are really

but a chapter in comparative physiology. Added to the different methods of research are the anatomic variations in the animals employed. The rabbit, for instance, differs in certain details from the dog and cat; and these latter two again present differences. The ophthalmic ganglion of the dog is of the mixed type (cells of the spinal and of the sympathetic type), whereas in the cat we have cells of the sympathetic type only. This is stated for purposes of illustration only, as up to the present it seems demonstrated, contrary to Nicati's belief, that the ciliary ganglion is without influence on the ophthalmotonus. Of course, there is a general anatomic scheme which holds good for all mammalia with certain variations in the species; a good example of this is the sympathetic system.

According to many authors there seem to be two nerves only which have to do with the ocular tension: the trigeminus and the sympathetic.

The ophthalmic branch of the *trigeminus* nerve sends nerve fibers to the eye either by the direct route of the long ciliary nerves, or through the indirect route of the short ciliary nerves. These latter are the efferent branches of the ophthalmic ganglion. We also know that those fibers of the fifth nerve which pierce the long root of this center pass thru it without giving off filaments, at least the purely sensitive ones do. Doyon, Morat, and F. Frank have studied the action of the trigeminus by working on the ophthalmic nerve close to the Gasserian ganglion, or on the trunk of the trigeminus where it leaves the bulb. In the dog they have shown that section produces hypotonus, irritation hypertonus.

In these experiments the essentially sensitive trigeminus is only indirectly concerned; the results obtained are in reality due to the sympathetic filaments which it contains, these filaments being vasodilators and vasoconstrictors, as proved by the following experiment. Exciting the trigeminus or the sympathetic in dog or cat raises the ocular tension, and with the ophthalmoscope one can see the dilated retinal vessels. If one starts with sec-

tioning the trigeminus, the exciting of the sympathetic of the same side has no effect on the ocular tension. If, for purpose of control, the sympathetic of the other side is excited we have vasodilatation and rise of tonus in the control eye.

The analysis of these two experiments shows us that the ophthalmic nerve, if it contains sympathetic filaments, has those of vasodilating nature. By sectioning the trigeminus or its branch, the ophthalmic nerve, these vasodilators are put out of commission, and the vasoconstrictors become predominant, with the result of lowered eye tension due to lessened volume of blood. Thru the studies of Doyon, Morat, Dastre and F. Frank we know that the sympathetic filaments reach the trigeminus either by way of the bulb (vertebral nerve of B. Frank), or by way of the sympathico-gasserian anastomosis. The part played by the trigeminus in the regulation of ocular tension becomes, therefore, nil, and the only nerve concerned is the sympathetic.

The *sympathetic* cervico-thoracic portion does not send an autonomous nerve to the eye; the sympathetic fibers come from the vertebral nerve and from the superior cervical ganglion. Those from the vertebral nerve come by the bulbus route; which it leaves when it penetrates the trunk of the fifth nerve to accompany the ophthalmic nerve, and its nasal branch to penetrate the eye in company with the long ciliary nerves. Some of the fibers follow the long root which the nasal nerve gives off to the ophthalmic ganglion; and, passing over to the short ciliary nerves, reach the sclera with these latter. Of the fibers coming from the superior cervical ganglion, some pass into the gasserian ganglion thru F. Frank's sympathico-gasserian anastomosis, and into the ophthalmic nerve, where they join the fibers of bulbo-vertebral origin. Others leave the superior cervical ganglion and mix with the plexus surrounding the internal carotid. From this carotid plexus comes the sympathetic root of the ciliary ganglion. We do not know

whether this root gives off anything to the ganglion, or whether it simply passes thru it, to enter the globe in company with the short ciliary nerves. All we know is that this root contains, besides vasodilator filaments vasoconstrictor filaments, being the only one to contain both, as the ophthalmic nerve contains vasodilators only.

From these anatomic details with which we have become acquainted thru physiology we see that the sympathetic system of the eye is a singularly complicated one; and is made more complicated by the fact that the superior cervical ganglion is not an autonomous center for the filaments which leave it. Certain filaments simply pass thru it, after being received from the cervical portion of which it is the upper extreme part. The same may be said of the vertebral nerve, which is

formed by the *rami communicantes* of the last cervical and the first dorsal roots. It is, therefore, in the cervico-thoracic medulla that we must look for the primary vasomotor centers of the head and eyeballs.

We have thus many points of attack in our experiments. To take as an example of the effect on the eyes, recall that section of the cervical sympathetic brings on exophthalmos, miosis, and (perhaps) a modification of the curvature of the lens; whereas, the irritation of the same nerve gives rise to the opposite, namely the syndrome of Basedow. The study of the relation of the sympathetic to ocular tension goes back to 1866, and the investigators included some of the best known in ocular physiology. To show the diversity of their findings the following table will be of interest:

DIVERSE RESULTS OF EXPERIMENTS.

- | | |
|--------------------------------------------------------|-----------------------------------------------------------------------------------------------------------------------------------------------------|
| (a) Section of the cervical sympathetic. | No effect (Grunhagen, Hippel).
Diminished tension (Neuschuler, Hertel).
Rise in tension and dilatation of the retinal vessels (Doyon, Morat). |
| (b) Irritation of the cervical sympathetic | No effect (Grunhagen and Hippel).
Slight rise of tension (Wegner).
Lowered tension even after irritation had ceased (Schulten, Doyon, Morat). |
| Experiments on Rabbit. | |
| (c) Irritation of the superior cervical ganglion | Increase of tension (Doyon and Morat). |
| (d) Irritation of the thoracic sympathetic | Lowered tension (Grunhagen, Hippel). |
| (e) Irritation of the carotid filament... | No effect (Angelucci). |
| Experiments on the Dog, or Cat. | |
| (a) Section of the cervical sympathetic. | No effect (Grunhagen and Hippel).
Lowered tension (Petit, Donders, Adamuck, Hertel, Neuschuler). |
| (b) Section of the upper cervical ganglion | Increased tension (Grunhagen, Hippel).
Lowered tension (Wegner, Dimitrowski, Angelucci, Schulten). |
| (c) Irritation of the cervical sympathetic | Increase of tension and of retinal circulation, production of Basedow's syndrome (Doyon and Morat). |
| (d) Irritation of the superior cervical ganglion | Increase of tension followed by a fall and finally hypotension (Grunhagen and Hippel, Bellarminoff, Graser, Henderson and Starling). |

Add to this that Lodato after passing a silk thread thru the superior cervical ganglion asserts that he observed lasting hypertonus due to the presence of this foreign body.

The contradictions in the foregoing table are more apparent than real. If the experiments are conducted with good instruments, a rational technic, sufficiently prolonged observation, there will be concordance of the final results. The differences are transitory only, and can often be accounted for by inequalities in technic and in the sensibility of the animals used.

IRRITATION OF THE CERVICAL SYMPATHETIC.

In a curarized rabbit, on the opposite side the registered blood pressure in the carotid shows no variation during the experiment. By Faradic irritation of the sympathetic during 30 to 120 seconds with a current sufficiently strong to induce mydriasis of the same side, in 16 cases Wessely obtained identical results, a constant hypotension. This begins a few seconds after irritation is begun; when this ceases the maximal drop is attained and persists for some time, and the tension does not return to its original level. As the ocular tension falls the oscillations of the ocular pulse disappear.

In a curarized dog or cat, the registered blood pressure or the carotid on the opposite side shows a slight rise in the cat, and a strong one in the dog. The tension, after an increase of 4 to 6 mm. Hg., drops rapidly and remains below normal during the time the current is passing. When the current is stopped the tension returns slowly to the initial point, and the ocular pulsations, which had disappeared, again become manifest.

The explanation of this difference in behavior at the beginning of the experiments is that the increase in tension is due to a venous stasis which causes a protrusion of the eyeball. As this stasis is less pronounced in the rabbit than in the cat or dog the former shows a drop only; whereas there is a momentary rise in the latter animals. As to the presence of a silk thread in the superior cervical ganglion causing hypertonus (Lodato), that experiment will not stand a rigid in-

vestigation, as the result here is not due to irritation but to a degeneration of the ganglion, which places it on a par with the result of resection of the sympathetic. As to the dilatation of the retinal vessels, this can be explained by anatomic variations. Wessely thinks that the cat has one innervation for the choroidal, and another for the retinal vessels. This need not astonish us when we reflect that the ciliary ganglion of the cat is anatomically different from that of the rabbit. Furthermore, we have proven that irritation of the sympathetic produces greater results in the rabbit than in the cat or dog, and we must recall the difficulty of producing cephalic stasis in the rabbit on account of the ample supply of blood from the vertebral region. In similar ways many of the apparent contradictions may be explained.

SECTION OF THE SPINAL SYMPATHETIC.

With the curarized rabbit (Wessely), blood pressure in the carotid on the opposite side shows negligible variations. After section of the sympathetic on the left side, after one or two minutes a rise in tension of left eye of 18 to 20 mm. Hg. occurs. The tension of the right eye shows a slight drop of 1 mm. Hg. Five minutes later the tension of the left eye begins to drop, and has reached the initial tension 15 minutes later. On the other hand, pulse and respiration have increased. Going back to the classic experiment of Claude Bernard, showing that section of the sympathetic causes a marked dilatation of all the vessels of the head, the only thing that surprises us is that the hypertension of the eye should not be more pronounced. Here we encounter the autoregulating device of the eye, which we considered when we were studying the relation between general blood pressure and ocular tension.

SYMPATHETIC CENTERS AND OCULAR VASOMOTOR NERVES.

The vasomotor nerves have often been described as valves which regulate the flow of blood, and the blood pressure in the different organs. This mechanism is under the control of centers of the first, second and third order.

A center of the first order is the bulbar center which lies on either side of the median line, at the level of the floor of the fourth ventricle. If this region is irritated electrically there is a general increase in the arterial pressure; if it is destroyed we have the opposite result. The fibers coming from this center descend along the anterolateral columns of the spinal cord, in which there exist, probably, accessory centers. Cutting the cord from below upwards brings on successively vascular paralysis of the region corresponding to the segment; and, vice versa, electric irritation of a segment of the cord produces elevation of the blood pressure of the region innervated by this segment. The sensitive impressions are conveyed both to the cord and the bulbus; and their role is the more clearly understood if we consider that they constitute the summit of the reflex arc.

The centers of the second order are the thoracic or cervico-cephalic ganglia. This is what makes the superior cervical ganglion important, the regulation of the ocular circulation. Thru their influence these centers can, up to a certain point, compensate for the experimental suppression of the bulbo-medullary centers. If to the mutilation of the latter, we add the destruction of the former, the result will be magnified. This explains why the experimenters on the influence of the sympathetic on the ocular tension have obtained more marked results from the destruction of the ganglion, than they have from the simple section of the cervical chain.

The centers of the third class are the peripheral centers situated in the organs themselves. Under the heading "anatomic details" it was shown how rich the choroid is in nerve cells (H. Müller's ganglion). This organ is particularly important for the eyeball, and its action manifests itself under widely varying conditions. For instance, it asserts itself when the superior cervical ganglion is resected, the effect of which it partially neutralizes. It is a well-known fact that the phenomena constituting the syndrome of Pourfour Du-

petit, falsely called the syndrome of Horner, are not permanent, but tend to disappear after several weeks.

In the experiments of Henderson and Starling, and of Wessely (injection of adrenalin into the vein), it is this organ which prevents the considerable elevation of blood pressure from causing a parallel rise in the ocular tension. It is this same organ which moderates the effect on the eye which we have described as produced by compression of the jugulars, or compensates the results of cutting the vagospinal or the cervical sympathetic. Even more evident is its action in the local inflammation produced by the subconjunctival injection of a strong solution of salt, bichloride of mercury, and similar fluids; or the irritations due to the experimental introduction of foreign bodies into the anterior chamber. In the latter case the result is often the inverse, namely vasodilatation and hypertonus.

From the sympathetic centers issue constricting and dilating fibers, which are not, strictly considered, antagonists. The constrictors are tonus producers, whose action can be readily understood when we consider that the vessels possess a circular musculature; and that a reduction of the caliber of the vessels is brought about by the action of the constrictors, with a resultant rise in the blood pressure above the point of action, and a corresponding fall of pressure in the veins below the point. The vasodilators, on the contrary, can have no direct action in the vessels, inasmuch as the vessel walls do not contain longitudinal muscles. Being without direct influence in the musculature their sphere of action lies in their influence on the constrictor nerves. This explains the lack of effect if one sections the dilators, and the intense effect of stimulating these same dilators.

This effect is naturally much greater when the vessels are contracted than on vessels already dilated; which explains the minimal result when one tries to increase the tension in an eye in which the tonus is already high, or,

in other words, in which the choroidal vessels are already dilated.

It would seem, therefore, that the dilators act by paralyzing temporarily the constrictors. In a general way the cervical sympathetic and, current opinion notwithstanding, the vasoconstricting filaments for the eyeball predominate over the dilators. This predominance explains the fact that irritation of the cervical sympathetic brings on a lowering of tension, out of all proportion to that caused by simply cutting the nerve. In the eye we must take into account the vasomotors which emanate from the nerve cells of the choroid; or, in other words, from the peripheral center. It is safe to assume that, altho the eye receives the greater part of its constrictors from the cervical ganglion, it is from the choroidal ganglion that arise the major part of the dilators. It is in this way that the hyperemia due to physical and chemical excitants (cauterization of the limbus, electrolysis of the angle, subconjunctival injections of sublimate, etc.), which are used to produce an artificial rise of tension is brought about.

AQUEOUS HUMOR AND TENSION.

Here we come to the kernel of the matter, and in this chapter will be found many heresies for those who believe in the classic theories of Leber, Ulrich, Priestley Smith, and others, which accept a current in the anterior chamber, and an angle of filtration, to which they cling as tho they were old friends. Very seductive, indeed, is the hypothesis of perpetual secretion of aqueous humor regulating the tension, very plausible, this outlet thru Schlemm's canal, a valve, as it were, the choking of which is fraught with the gravest consequences. Nothing more erroneous, when one studies and analyzes a little more closely.

Up to the present the subject of ocular tension and aqueous humor have been kept apart intentionally, and their perfect independence thus established. There is no link between them. Ophthalmotonus is a purely mechanical thing, inasmuch as it is regulated solely by the greater or less repletion

of the vessels of the uvea. The aqueous humor, as shown in a previous article, is a matter of histology and biology. Its relation to ocular tension lies in its bearing the consequences of experimental or pathologic vasodilatation, and nothing else. It acts as a buffer when the ocular tension passes beyond certain limits.

The aqueous humor which leaves the retina and fills the anterior chamber (the vitreous is nothing more nor less than retinal neuroglia), belongs to that class of liquids which might be designated as mineral sera. Very rich in NaCl, it is a splendid medium for maintaining the retinal cells in a state of normal excitability; but its poverty in protein elements deprives it of assuming a nutritive function. The duty of nutrition belongs, anyway, to the blood, with which the eyeball is so richly provided. The aqueous humor is not undergoing continuous secretion or dialyzation. On the contrary, it is a very sluggish stream, whose resorption takes place, without doubt, thru the veins, as is generally done thruout the organism. The details of this have been given in the *Annales d'Oculistique* for February, March and April, 1917. Here we shall take up a few points only.

After paracentesis of the anterior chamber we find this space rapidly refilled with a liquid, called aqueous humor of the "second formation." This new humor has not the composition of the original humor, but contains fibrin, albumin, and numerous other substances which abound in the serum of the blood. We have now to determine whether these substances remain in the new aqueous indefinitely, or whether an aqueous of original composition can be found in a globe that has once been tapped. It has been asserted that these foreign substances disappear from the aqueous with greater or less rapidity, and this is one of the arguments of the supporters of Leber's theory of a current in the anterior chamber. They claim that the disappearance of hyphemas and hypopions in an eye that has not been tapped is a further support of their theory.

But they are wrong, because: We see subcutaneous hematomata disappear without imputing their removal to a liquid current; we know that their disappearance is the work of cells, phagocyte, lymphatic and connective. In a recent article Leboucq has shown us how this takes place in the eye. If a drop of neutral sterile oil is brought into the anterior chamber we can watch its breaking up into fragments, and the penetration of these oily particles into the iris. When these particles reach the root of the iris they are swallowed by leucocytes, which carry them thru the scleral cup by way of the emissary veins. Once outside of the eye these droplets again become free, and one can see them agglomerate themselves in the lymphatics of the orbit. This proves that foreign substances can be eliminated from the aqueous without the elimination of the aqueous itself. (We cannot assume with Leboucq that the aqueous is lymph, as the chemical composition of the two fluids is entirely different).

The fluid which refills the drained anterior chamber is composed of the remainder of the aqueous which laved this veritable fibrillar sponge called the vitreous; and serum transuded from the capillaries distended by the fall in pressure caused by the puncture of the anterior chamber. The aqueous of "second formation" is, therefore, a mixture of normal aqueous, brought forward by the fall in pressure, and of a certain amount of blood serum. In proof of this we have: If, after death, we empty the anterior chamber, we see it refill. Deutschmann, who noted this fact a long time ago, concluded that this was the result of forward transudation of the vitreous. It cannot be fibrillary vitreous, but liquid vitreous that he has in mind.

We can suppress this phenomenon. If, before puncturing the anterior chamber we inject under the conjunctiva pure adrenalin, whose vasoconstricting power is very great, the anterior chamber will fill feebly and slowly with a small quantity of liquid whose composition is that of the normal aqueous. The process is, there-

fore, the same in the living and the dead subject.

The experiments of Ehrlich and his imitators, who injected fluorescein into the venous circulation, to see it reappear either thru the pupil or on the anterior surface of the iris, is another argument of the proponents of a continuous secretion of the aqueous humor. These phenomena have been generalized and incorrectly interpreted. It is true that one sees in the intact eye of the rabbit a green line at the level of the pupil, a few instants after the injection of fluorescein. But this is a phenomenon restricted to the rabbit, and does not manifest itself in man or monkey under normal conditions. There is a way, however, of provoking it in these latter, and that is by inducing hyperemia either thru an irritating subconjunctival injection, or thru mechanical irritation of the cornea. We must, therefore, conclude that the passage of fluorescein into the aqueous humor is due solely to the transudation of this substance thru the dilated capillaries; and, if in the rabbit it takes place in normal eyes, it can be explained by the greater permeability of the ocular vessels in the rabbit. Besides, the amount of fluorescein introduced into the vein must be considerable in proportion to the bodyweight.

Even conceding that the liquid reforming after puncture is a mixture of aqueous and blood serum, how shall we explain the rapid return of the eye to normal? The explanation is easily given. If we empty the anterior chamber on the cadaver we see the chamber refill almost completely, but the tension remains about 8 mm. Hg. below the anteoperative tension. If we repeat the experiment on the living the tension reestablishes itself, not alone thru transudation of serum, but also thru the repletion of the choroidal erectile system, which fills with blood and becomes turgescient. Should the vessels of the choroid have become friable thru sclerosis we may even get hemorrhages, (the explosive hemorrhages occurring during cataract extractions). This distension of the choroid is governed in part by a reflex vasodilatation,

but to this action of the sympathetic is added a passive dilatation due to the sudden fall of pressure caused by the evacuation of the fluid, and it is, therefore, the decompression which is the principal factor.

So far, in speaking of the influence of the general and of the local circulation on the ocular tension, no mention has been made of the effects of the experiments on the composition of the aqueous humor.

The methods of increasing blood pressure and ocular tension by manipulations at a distance, such as compression of the abdominal aorta, section of the pneumogastric, constriction of the neck, have already been mentioned. The results of these measures on the aqueous are quite variable. Section of the pneumogastric rarely changes the albumin content, (once in four times, Wessely); nor does compression of the aorta or constriction of the neck. This is explained by the slight rise in ocular tension, which rarely rises more than 6 or 7 mm. Hg. above the initial tension; so that the consequent intraocular vasodilatation and transudation of serum is inconsiderable. As already explained this resistance of the eye to an elevation of the cephalic blood pressure is due to the counter action of the sympathetic regulating system situated in the eye itself.

The intraocular tension may be indirectly increased by the section of the cervical sympathetic, which, according to Lodato, Mastrobuono and others, entails a notable increase in the amount of albumin. If we inject at the same time fluorescein into the veins of the animal, we shall find the coloring matter appear sooner on the operated side. This led Shöler and Uhthoff to the conclusion that the secretion was accelerated by the operation. It was, in reality, a question of intraocular vasodilatation.

One might, however, argue that certain experimenters have found a modification of the aqueous; and that in cases where it should not have manifested itself, for instance, after section or irritation of the cervical sympathetic

(or of the superior ganglion). The reader is asked to recall what has been said when on the subject of interventions on the sympathetic. He will remember that irritation of the nerve lowers the ocular tension in all animals; but that preceding the drop in the dog and the cat there is a preliminary sharp rise. This rise is due to the marked exophthalmus resulting from the irritation of the nerve; which, in turn is due to the orbital venous stasis, thus explaining the discrepancy between the results obtained by Lodato, Scalinci and others.

If before lowering the intraocular tension by faradisation of the sympathetic, we allow the aqueous to escape, we shall notice that the anterior chamber is very slow in refilling, and that it contains a fluid almost identical with the normal aqueous. In other words, we have obtained with electric irritation the same effect as that which we obtained by injecting adrenalin. This organic product influences the organic elements. The part of the blood pressure in the regeneration of the aqueous after ligation of a carotid shows itself by the slower refilling of the chamber, on the side on which the carotid has been ligated.

Measures applied to the globe itself are numerous, and bring about much more energetic changes in the ocular tension than the indirect methods. Thus we have seen that subconjunctival injections of a 10 per cent solution of NaCl. can cause a rise of 40 to 70 mm. Hg. While this rise in tension is taking place there is also a change in the aqueous, which reaches an albumin content of 0.9 to 1.0 per cent (normal 0.02 to 0.03).

A graphic chart of both values shows a surprising parallelism between the two. The rise is equally sharp, and almost synchronous. The drop, however, is much slower for the albumin, which is present some time after the tension has returned to normal. With the albumin the antibodies make their appearance. If an injection of fluorescein or uranin is made before the subconjunctival injection the passage of the coloring matter into the chamber

is so much accelerated and is much more intense on the irritated side.

Thus we find that the rise in tonus, the qualitative changes of the aqueous, and the appearance of the fluorescein show a remarkable parallelism, and there is no doubt that all these processes are set in play by the dilatation of the vessels of the uvea. They can all be slowed down, or prevented, by any measure which will cause anemia of the globe, such as faradisation of the sympathetic, intravenous injection of adrenalin, ligating the carotid of the same side, etc. It seems illogical to attribute the ocular hypertension to the increase in albuminoid substances in the aqueous; the proteid substances are present in virtue of a transudation of serum thru the dilated capillaries.

In an organ possessing an erectile tissue such as the eye possesses, it is not surprising that massage produces a certain lowering of the tension. The deviations are small, Knapp obtaining differences of 3 to 10 mm. Hg. only. The return to normal is very rapid, and qualitative changes are not noted. This same mechanism might be at the bottom of the changes noted with the tonometer of Schiötz by P. van Gelder. The repeated application of the instrument at intervals of 3 or 5 seconds has given figures inferior to the first ones. It is unnecessary to invoke an "active filtration of the aqueous humor" in explanation.

Numerous attempts have been made to secure artificially a lasting rise in the ocular tension. An excellent way, as already shown, is to induce choroidal stasis by supressing the anterior and posterior ciliary veins. In the dog the ligature of the anterior ciliary veins will even be attended with a rapid disorganization of the globe. Ligature of the vorticose veins will give rise to a marked stasis, with displacement of the ciliary region, the iris, and the crystalline lens.

These methods give rise to a hypertension by passive filling of the vessels above the point of constriction. There are other methods which give rise to an active vasodilatation by irritation of the nerve, such as strongly hypertonic

saline injections, sublimate, sodium citrat, sodium fluorid, etc. Add to these such procedures as cauterization of the limbus. Whereas purely conjunctival inflammations and those affecting the episclera only superficially do not lead to an increase of tension, and the conjunctival vasodilatation is accompanied by a slight decrease of tension; anything acting on the cornea, and especially on the limbus, is accompanied by a rise in tension, either thru reflex, or thru a mechanical impediment to the anterior venous circulation. On the other hand, we often see contusions of the eye with small iridic hemorrhages showing a decrease of tension, in spite of the presence of blood in the anterior chamber. This is accounted for by the reflex vasoconstriction due to shock. Other contusions of the eye may set in play the ocular vasodilating reflex, but the rise in tension escapes unless it is considerable. In these cases, also, we have hemorrhages from the iris and ciliary body; which are of importance as noted in a number of clinical cases.

The composition of the aqueous humor is a far more sensitive test. Often, were we allowed to withdraw it, we would find albumin in the aqueous. Even after repeated instillations of distilled water we find this proteid in the aqueous. Misled by the theories of Leber as to the outlets for the aqueous and the consequences of their obstruction, certain experimenters have been led into adventurous procedures, such as injecting paraffin, electrolytic iron, negrosin, oil of Scarlach, etc., into the anterior chamber. Only in rare instances could the disappearance of the canal of Schlemm, or the effacement of the iridocorneal angle be determined; as could easily have been prophesied, the microscope showed profound lesions of the iris and choroid.

Osmotic processes may be used to vary the ocular tension. In 1913 Hertel showed that a strong hypotonus followed the feeding to a rabbit, or the injecting into its veins, of large quantities of salt or sugar. The tension returns to normal when the water withdrawn from the ocular tissues has been restored; and this return to the normal

may be hastened by replacing the concentrated salt solution by a hypotonic solution.

If the greater or less repletion of the uveal tissue is the only thing which regulates ocular tension, how about the hypotension in cases of irido-cyclitis and detachment of the retina? So far no mention has been made of pathologic conditions; as there is a wide breach between experiments made on healthy tissues, and clinical observations on diseased tissues. It stands to reason that a vascular system which is more or less destroyed by an acute or chronic inflammation is not capable of proper function. This becomes more apparent when we reflect that many diseases produce arterial obliterations with consequent lessened blood supply.

COLLYRIA AND PHYSIOLOGIC OCULAR TENSION.

Nothing is more obscure or debatable than the action of the alkaloids, and the various substances employed as collyria, on the ocular tension. The great clinical interest attached to them has made them the object of much study. For instance, why does atropin cause hypertonus, and eserin also in certain cases of glaucoma? The main difficulty lies in the uncertainty of the physiologists as to the point of attack of most of the drugs. Since the time of Brown-Sequard there has been a continuous discussion as to whether atropin acts on the terminal nerves or the muscular fibers; and the remarkable history of the antagonists does not shed any light on the controversy. The reasoning has been somewhat as follows: "An antagonist of atropin seems to exert its action on a well defined anatomic element; therefore, atropin acts on the same element. Or, atropin increases the action of some other alkaloid, which has an action similar to atropin, therefore both act on distinct elements. In many of these cases the conclusion is a *petitio principii*, inasmuch as the point of attack of atropin is still under discussion," (Nuel, *Diction. de Phys. de Richet*).

Animals act differently from man, and again differently according to species. For example: The instillation of

a 1 per cent solution of eserin into the conjunctival sac of a rabbit has a very insignificant action on the blood pressure. Whereas the effect on a cat is so great that this animal cannot be used in experiments on the ocular tension. The same is the case with atropin. Man is extremely susceptible to this alkaloid, the monkey, dog and cat much less; and still less the rabbit, guinea pig, and horse. Rabbits, goats, sheep and pigs eat belladonna leaves with impunity.

The investigations into the untoward action of certain collyria in pathologic hypertension have been without result, for the reason that we are unable to set up in animals affections similar to glaucoma in man. The hypertension which we can provoke has but a far-fetched relation to the real condition. It demonstrates certain mechanisms; but, as it is accidental and transitory, it can hardly serve as a basis for further researches on the action of the collyria. There are still to be considered the normal human eye and tonometric studies. But the action of the collyria on the normal eye is too weak to be measured with our clinical instruments which allow the taking of data at greater or less intervals only, whereas we should have a constant application of one hour at least with graphic registration. Notwithstanding, we shall see what has been done and said about the chief drugs used.

Atropin.—Experiments on the normal eyes of man and animals has led to three different opinions. Some hold that it induces a lowering of the ocular tension. Dor, experimenting on man, found a lowering of the tension in 20 per cent, a rise of tension in 3 per cent, and no effect in 16 per cent of his cases. He thought that the repeated instillation of atropin during several consecutive days resulted in a lowering of the tension which was more manifest if the other eye had been under the influence of eserin. Pflüger estimated the lowering of the tension in man at 80 per cent, and he found the same figure when experimenting with the manometer on the rabbit. Golovine, using the tonometer of Malakoff, found this

hypotension accompanied by a corneal lesion, and preceded by a slight hypertension. This hypotonising action was so generally admitted that von Graefe thought its use indicated in those corneal lesions attended by "edema of the parenchyma." Others, such as Laqueur, Weber, Stocker, etc., were of the opinion that atropin increases the ocular tension. Weber held that it had a different action on the vitreous than on the aqueous, an assertion which was sharply criticised by Schulten, and led to entoptic studies to prove that the vitreous was not influenced by either atropin or eserine. Others, again, such as Schiötz, Isakowitz, Langenhan, etc., think that atropin has no effect in the healthy eye. In view of this diversity of opinion it is rather difficult to give a satisfactory explanation of the hypertensor character of atropin in the glaucomas. The idea that it might have an influence by activating "the secretion" of the aqueous is controverted not alone by what has already been said, but also by the known fact that this alkaloid has the well known property of suspending the glandular secretions.

On the other hand, the ocular tension seeming to be under the primordial influence of the local circulation, it is interesting to determine the action of atropin on the blood vessels. Now, according to the dose, atropin diminishes and then suppresses the constricting action of the vasomotors. The normal tonus of the small vessels relaxes and the vessels dilate. Wegener is of the opinion, that as the capillaries do not offer any resistance, the blood passes into the larger canals and the tension falls. While this may be true for the blood pressure, is it also true for the ocular tension?

If we admit that the choroid is distended with blood its increase in size should logically increase the ocular tension, inasmuch as the "humors" of the globe are incompressible. But, the arterial pressure being low, the increase in volume of the erectile uveal tissue must be inconsiderable, and the choroid all the less distended in that it is held in check on the outer side by

the sclera, and on the inside by the pressure inherent in the vitreous and aqueous which lave the choroid. Thus we may understand the small effect of atropin on the healthy eye, in spite of its role of dilator inherent to its attack either on the vasomotor nerves, or on the contractile element itself. We must also keep account of the size of the doses used and of the sensibility of the organism on which we are experimenting. To account for the decided hypotonising effect in many cases of glaucoma we need but point to the vascular lesions, especially those of the veins; which, by impeding the return flow, bring on an increase of blood pressure all the greater proximally as the atropin paralysis of the arteries allows the choroid to assume a marked distension.

Eserine.—Like atropin and adrenalin a 1 per cent collyrium of eserine has a decided action on the pupil. That this is local is proved by the action taking place in an enucleated eye also. The action of eserine on the tension of the healthy globe is, however, but slight. In an eye with pathologic hypertension eserine will cause a drop in tension; altho there are many exceptions to this rule, the explanation for which was sought in experiments on animals and man. Man is particularly susceptible to eserine; then come dog and cat, with the rabbit bringing up the rear.

In man it was first held, following Laqueur, that the effect was nil. Later, the use of the tonometer of Schiötz having been popularized, many authors claimed that the tension, after a slight elevation, suffered a decline oscillating between 2 and 5 mm. Hg. This decline gave way to normal tension after 24 hours. Such was the opinion held by Pflueger in 1882. Experiments on the cat do not seem to give the same results. A lowering of the tension of 5 to 10 mm. Hg. is produced by a few drops of eserine in 4 to 9 minutes, after which there is an increase in tension (Lewin and Guillery). Wessely holds that the cat is particularly responsive to eserine; he thinks that even in infrequent doses the diffusion thru the cat's organism is rapid, and brings on a low-

ering of the arterial pressure which vitiates the manometric tracings of the ocular tension.

On the rabbit, on the contrary, the results are uniform. Wessely has furnished us charts showing that the tension after an initial rise of ten to fifteen minutes after the instillation, immediately after the appearance of myosis, which reaches 15 to 17 mm. Hg. above the normal, drops gradually to reach the point of departure in an hour. It may even keep on dropping, which may be accounted for by the curarization, although the rapidity with which it drops would make it seem due to the action of the eserine. Keeping the proportions, and remembering that the tonometer does not possess the exactness of the manometer, we believe that we find the same behavior in the human eye.

Eserine is a vasoconstrictor, of which fact one can convince himself by watching with a loupe the contraction of the vessels in a hyperemic conjunctiva or a corneal pannus. This contraction lasts for about fifteen minutes, without being followed by a marked dilatation. The inspection of the deeper vessels is not easy. Wessely has noticed, not a constriction, but a dilatation of the vessels of the iris and ciliary body. He finds a striking similarity between the curves of the ocular tension after instilling eserine and those obtained after injecting solutions of NaCl. under the conjunctiva, and gives them both the designation of curves of "hyperemia of reaction."

Acting on the supposition that the eserine hypertension might be due to vasodilatation, he analyzed the aqueous and found a marked increase in its albumin content. He then injected fluorescein, to which the rabbit is highly susceptible, and found that on the eserinated eye the line of Ehrlich showed much more promptly than in the fellow eye. As both phenomena proved the passage of serum thru the vessels, he undertook to prove the opposite by diminishing the permeability of the vessels by injecting calcium salts,

which was followed by a diminution of the albumin content.

This leads us to the contemplation of the questions whether eserine has the same action on the human eye as on the eye of the rabbit; whether eserine acts differently on the external and on the internal vessels, and whether Weber was right in asserting that eserine caused a lowering of the pressure in the anterior chamber, and an elevation of pressure in the vitreous. We might repeat concerning eserine what has already been said about atropine as regards the point of attack. How does eserine determine a constriction of the pupil? Is the cramp due to its acting on the nonstriated muscle, or to a suspension of the sympathetic influence (the sympathetic is a dilator)? This problem might have a certain interest for the ophthalmotonus, inasmuch as the sympathetic system and the vasomotors are interdependent.

The sympathetic does not, however, seem to be paralyzed, for, even in the eyes of dogs and rabbits which have been strongly eserinated, the irritation of the cervical sympathetic will still show a reaction on the pupil. We can see the same thing if after dropping eserine in both eyes, we cut the sympathetic on one side, when we shall find the pupil narrower on the cut side. Even after animals have been slowly poisoned to death with calabar the sympathetic remains irritable. It is, therefore, probable that eserine acts on the muscular fiber. It produces a spasm of the sphincter, and, according to dose and circumstance, a spasm of the vessels. This will account for the hypotensor character which is now accorded it, which is not very prominent in the normal eye.

Pilocarpin.—Some authors hold with Rollet in denying pilocarpin all action on the tension of the normal eye. Others, with Heilbrun, admit in man a slight rise, as was the case with eserine; the curve being more marked and more regular than it is with eserine. Pflüger and Golovin could find no relation between the variations of tension and the state of the pupil in their experiments on animals.

Cocain.—There are some authors, who, like Rollet, accord the role of hypertensor to cocain; instillation of a 2 per cent solution of the rabbit is supposed to produce a slight elevation of tension, it being undetermined whether this is followed by a decrease. On the other hand, renewed instillations cause hypotension thru corneal lesions (?). More recent experimenters, using the tonometer of Schiötz, have found in man after instilling a 3 per cent solution a hypotonic effect, some of 5 mm. Hg., others from 1 to 4. This is perfectly plausible if we reflect that the vasoconstricting effect of this alkaloid becomes the more marked the stronger the solution employed. This is why its use may delay the reestablishment of the anterior chamber after a cataract extraction, and give rise to prolapse of the cornea in persons whose vessels are modified by age. Fourière (1913) thinks that the action of cocain on the normal eye is variable, sometimes producing a light hypertension which is transitory, sometimes the opposite, sometimes having no effect whatsoever.

Novocain and holocain seem without influence on the ocular tension.

Adrenalin.—Rubert found that after instilling a fresh solution of 1 to 1,000 of adrenalin there was an initial hypotension, succeeded by a hypertension; which again gave way to a second and last hypotension. The results which differ from this seem to be due to differences in the drug used. Synthetic adrenalin has not the same effect as the natural product, the adrenalin of Hoechst giving just the opposite results. Adrenalin acts on the muscles which are innervated by the sympathetic.

Dionin.—This drug produces a nar-

rowing of the pupil of varying duration, which in most cases is followed by a dilatation. The myosis follows closely on the chemosis produced by the drug. The tension begins to rise after the instillation, reaches a point several degrees above, and, after returning to normal, sometimes drops below it (Toizyski).

It seems useless to continue with the enumeration of drugs the action of which on the ocular tension has been but vaguely determined. Without receding from our expressed opinion concerning the difficulty of interpreting the data of these experiments, we may profit from well known facts, and these are:

(a) The tension of the normal eye is but little influenced by collyria;

(b) The variations in tension are not dependent on the contraction or dilatation of the pupil;

(c) The action on the vessels seems to be the most important thing. Vaso-dilatation means a slight rise in pressure, vasoconstriction the opposite. This effect may be masked by the secondary action of the drug on the neighboring tissues and, particularly, on the nervous system. Not enough stress is laid on the size of the dose, the sensibility of the subject, and, most especially, on the patulence of the vessels at the moment of instillation.

In closing, it is of interest to note that nitrite of amyl, caffein, and antipyrin, taken by mouth, increase the ocular tension. The point is, that altho these drugs lessen the arterial blood pressure in other parts, they cause a dilatation of the cerebral blood vessels. Knowing the similarity existing between the innervation and the circulation in the brain and the eyeball these facts seem significant.

SHORT ABSTRACTS.

Important points capable of brief presentation are here noticed. The systematic review of current literature is to be found in the Digest of the Literature.

A. A. Gruenbaum.—Psychophysics of Optical Fatigue.—*Tijdschr. v. Geneesk.*, January 27, 1917.

The method of the continual increase of the frequency of the flickering light allows a very accurate determination of the corresponding threshold values, that is the number of interruptions per second, when the flickering light just produces a constant impression. These threshold values are dependent on the degree of fatigue, which strong stimulation produces. If these threshold determinations are made during different times after fatigue the degree of fatigue and the gradually appearing restoration can be expressed in the curves of the increasing threshold values.

The course of these curves depends on the fatigue duration, and the intensity of the flickering light; and shows over compensation toward the return to the norm, with the direct fatigue of the exposed eye (distinguishing of the single light stimuli with higher frequency than in the norm). If one eye is examined, while the other one is exposed to the fatigue excitation, the curve shows the same curve, but without phenomena of supercompensation. Therefore a consensual fatigue of the eye exists and the reactions of the latter over compensation are characteristic for the restitution of the peripheric sensitive substances.

The comparison of the binocular and monocular fatigue leads to the conclusion, that binocular summation of the fatigue reactions does not exist.

E. E. B.

Bichon.—Colloidal Sulphur in Rheumatic Ocular Disease.—(*La Clinique Ophthalmologique*, July, 1917.) Arthritis combined with syphilis is one of the most frequent causes of inflammatory processes of the iris and sclera affecting often both eyes. It may come during or following the acute joint con-

dition. Up to the present the salicylates and aspirin have been used, but these drugs disturb the digestion and are too frequently followed by recurrences. Loeper obtained excellent results in rheumatic affections with colloidal sulphur. The method of choice is intravenous but they may be administered intramuscularly. Each ampule contains 2 cc. and is given every day or every second day. A series is ten to twelve injections. Five observations from the army are published in detail. In none of the cases has pain or any other symptoms followed the injections. A complete cure was effected in every case in a much shorter period than is necessary by the old time means.

J. S. W.

Morax, V.—Parinaud's Conjunctivitis.—(*Brit. Jour. Ophth.*, March, 1918.) The writer gives a brief account of researches made at the Lariboisière Hospital which, although they have not explained the nature of the infective agent in Parinaud's conjunctivitis, prove that this disease and tuberculous conjunctivitis, so often confused, are distinct morbid entities and in no way related to one another.

Incidentally he shows that Goldzieher was not the first to describe Parinaud's conjunctivitis; the case published in 1882 and two others reported in 1893, though they had a few clinical features in common, were shown by the histologic examination to be examples of the follicular form of tuberculous conjunctivitis without ulceration, since giant cells were demonstrated in the middle of the infiltrated nodules; this finding sufficiently differentiated even though inoculations on guinea-pigs were omitted.

Parinaud was well acquainted with tuberculous conjunctivitis, and never failed to have inoculations made when-

ever he suspected the tuberculous nature of a lesion. In a series of cases published by his former pupils, inoculations on guinea-pigs with a piece of conjunctival tissue, or of pus derived from infected glands, gave negative results, and the histologic examination of the conjunctiva never revealed the presence of giant cells; whereas in tuberculous conjunctivitis the presence of these structures was constant. And so also was the positive result obtained by inoculations on guinea-pigs with a piece of the conjunctiva or with pus, either subcutaneously or intra-peritoneally. A differential diagnosis between Parinaud's conjunctivitis and tuberculous conjunctivitis, by their clinical features alone, is often a delicate procedure. The error is much the more common as it is customary to say that tuberculous conjunctivitis is always accompanied by ulcerations. Cases, however, where we fail to notice any ulceration are by no means exceptional, and these are precisely the cases that are mistaken for Parinaud's conjunctivitis.

It is always easy to cut out a small piece of the conjunctiva, half of which is used for inoculation and culture, and the remaining half for histologic examination. If the histologic examination shows giant cells, we have to deal either with a case of tuberculosis or with one of sporotrichosis. In the first case inoculation will clear up the diagnosis, and in the second, a culture on usual media will show the typical cultural characters of the fungus. If the slide does not show giant cells, the differential diagnosis will have to be made between Parinaud's conjunctivitis and specific disease. In such a case experimental inoculation will prove negative. Microscopic examination may or may not show the spirocheta pallida, and even in cases of undoubted syphilis, we

must say, it is not always possible to find the infective organism.

Morax points out that up to now we have not been able to discover the infective agent of Parinaud's disease—but this is unfortunately true of a number of other diseases. We must admit that the clinical features by themselves are not always sufficient to clear up differential diagnosis, and we should rather trust the experimental or histologic examination whether we meet with a conjunctival affection with involvement of the lymphatic glands. The diagnosis of Parinaud's conjunctivitis should be reserved for those cases where the absence of giant cells, and the negative result of inoculation and culture would justify the elimination of tuberculosis, sporotrichosis, etc., of the conjunctiva.

C. H. M.

Fernandez de Castro.—Parinaud's Conjunctivitis, Tuberculosis of the Conjunctiva. (Bull. de la Soc. de Oft. de Buenos Aires, v. 4, p. 80.)

History of a case of Parinaud's conjunctivitis with red and yellow granulations and ulcers in both lids, accompanied by swelling of the parotid and preauricular glands. The frosts of the discharge and the inoculation of a piece of conjunctiva under the cellular tissue of the abdomen of a guinea-pig proved negative. Pus from the preauricular gland was sterile. However, histologic examination of a piece of conjunctiva near one ulcer showed zones of necrosis and cells of Langhans, indicative of tubercular infection. Von Pirquet reaction was strongly positive.

A new inoculation of conjunctiva under the skin in a guinea-pig produced caseous glands in the groin.

The author considers his case one of undoubted tubercular origin.

U. T.

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OCULAR LESIONS FROM FILARIA AND ALLIED PARASITES.

The round worm and thread worm have been known as intestinal parasites for many centuries; and the *Filaria Loa*, as a parasite infecting the eye, since 1778. Yet apparently this class of Nematoda or round worms, constitute one of the most promising fields for investigation. Many isolated observations have been made, but uncertainty and confusion still prevail with regard to all but a few specific forms. Even the latest contribution to the subject, that of Pacheco Luna, (p. 122), while extending the range of interesting facts regarding filariasis, cannot be said to make our knowledge more certain, or to bring much advance in it in the direction of order.

A parasitic disease comes to be well understood only when its cause is known. The discovery of a causative organism, life history and habitat, has revolutionized our knowledge of every disease for which this has been accomplished; not only our theory of it; but equally its diagnosis and treatment. This has been as true of diseases studied for thousands of years, like

Malaria and Tuberculosis, as it has of Blastomycosis or Infective Jaundice. Even from the standpoint of the practicing physician, the exact description of the cause of a disease will outweigh in value the most elaborate clinical reports or refinements of diagnosis.

This is particularly applicable to our knowledge of filarial disease, with regard to which there has been a large accumulation of incomplete accounts, that throw but little real light upon the subject. Ward classes as "*Agamofilaria*," sexually immature, a large number of reported cases in which the incompleteness of the description, or the immaturity of the specimens, makes it impossible to determine the species to which they belong. Serious confusion has arisen both with regard to the species that affect the eye, and the regions in which they are endemic.

The Filaria Loa, recorded 140 years ago as infecting the eye, is the best known of these parasites. The first six cases were reported from the West Indies and from the Northern Coast of South America; and five of the six were in negroes who had been imported from the West Coast of Africa.

Ward points out that no case has been reported from the West Indies since 1845. It may be assumed that this disappearance of the disease was connected with the suppression of the African slave trade. The subsequent cases reported from America, beginning with that reported by Wilson to the American Ophthalmological Society in 1890, have occurred in persons who had resided in Africa, especially in the region of the Congo, mostly as missionaries. It can now be regarded as settled that this parasite enters its host only in West Africa. But, living in the tissues for many years, it is liable to be carried to any part of the world; where, however, it does not find the conditions necessary for its transmission.

These worms are nearly always multiple. At autopsy many have been discovered in the superficial connective tissue, with none in the head or region of the eye. As a rule, only the immature forms are found in the region of the eye, the fully developed worm showing little tendency to migrate. The former may move rapidly under the conjunctiva or the thin skin of the lids; but their presence here is quite unusual and erratic. They are more frequently felt in other parts, but much of the time give rise to no sensation whatever.

The presence of a worm about the eye may cause a stinging, pricking, or boring sensation, or the feeling of a foreign body; with conjunctival redness which quickly subsides when the parasite moves elsewhere. In other parts it may cause some soreness or tenderness. Or there may be swelling localized or diffuse; which may look like cellulitis, but usually disappears in a day or two. When visible in or near the eye, the removal of the worm has not been difficult, and it has been killed by an injection of mercuric chlorid.

Filaria bancrofti is widely distributed in tropical regions. In Samoa 60 per cent of the inhabitants show marks of the disease and probably all the natives are infected sometime during life. The

larval form, infesting the blood at night, was given the name *Microfilaria nocturna*; in contradistinction to *Microfilaria diurna*, the larva of the *Filaria Loa*, found in the blood by day. Filarial elephantiasis, caused by blocking of the lymph channels has long been known. Such blocking of lymph channels if temporary may cause edema of the lids; and recurring attacks may result in a condition resembling blepharo chalcasis. Pterygium is extremely common in Samoa, and A. Leber has demonstrated filaria in these growths. He also observed evidences of obstruction of the circulation in the choroid and retina by miliary thrombi, causing hemorrhages and areas of degeneration.

Numerous cases of what were supposed to be other forms of filarial disease have been described. But probably most of them were due to forms of filaria that usually infest the eyes of the lower animals, and have accidentally gained admission to a human host. The large number of species already known to infest the lower animals gives some idea of the wide extent of this field. On the other hand the Guinea worm, *Dracunculus medienensis*, has been supposed to cause ocular lesions, because it was confused with the *Filaria Loa*. An instance of an allied form, commonly belonging in the eyes of other animals, is that of a species recently reported from China, as found both in human eyes and those of a dog. The specimens were regarded by Houghton as similar to those previously described under different names, *Filaria palpebraris*, of Wilson; and found in horses and cattle in Europe and India.

The cases in which filaria were actually found within the eyeball, in the anterior chamber by Barkan, and in the cataractous lens by the elder Graefe and Geschiedt, all belong in the category of unknown species. All seem to have arisen outside the tropical habitat of the *loa* and *bancrofti*. The description of a supposed filarial disease affecting the conjunctiva in children in Mexico and Central America, by Eisen, varies widely from most descriptions of filarial disease, and is not definite as regards the

organism. The clinical conditions to which attention is called by Pacheco Luna might readily be assigned to the *bancrofti* or some closely related form. In future much of the uncertainty of this subject can be avoided if the worms found are in each case sent to some expert zoologist. Ward points out that they can be permanently preserved by placing in 70 to 85 per cent alcohol, to which has been added 5 or 10 per cent of glycerin.

In the same order with the filaria—the Nematoda or true worms—belong the Ascaroidea. One of these, the round worm of the horse, *Ascaris megalcephala*, often examined in laboratories, produces extremely unpleasant and disabling symptoms in workers who are susceptible to it. These include irritation of the conjunctiva, with itching of the lids and caruncles that induce rubbing, which brings on severe swelling of the parts. It would seem that extreme susceptibility to this irritant may account for those cases of conjunctivitis which arise from being near horses. Dorff has experimented with regard to the irritant effects of the *ascaris*, and finds that they are exerted on the walls of the blood vessels, and are neutralized by adrenalin.

Among the nematode worms we also find the hookworms. *Ancylostoma duodenale* and *Necator americanus*. The eye symptoms that accompany hookworm disease have been regarded as due to the anemia; but there is evidence that they are directly connected with the toxins of the worm. Bietti reported amblyopia and central scotoma in hookworm disease, and Inouye, a case of retrobulbar neuritis, cured in ten days by thymol treatment. Calhoun regards the cataract as connected with the toxemia.

The ocular lesions produced by nematode worms offer a great field for accurate scientific observation. This is established by the many suggestive papers already to be found in the literature. More exact detailed descriptions, both of symptoms and parasites, with laboratory studies of the organisms and their products, will bring rich returns for the labor expended.

E. J.

BOOK NOTICES.

Atlas d'Ophthalmoscopie de Guerre, par **Le Professeur Felix Lagrange** de Bordeaux. Large 8vo, pages 255, with 100 plates, 20 in colors, and 12 diagrams in the text. Paris, Masson et Cie, 1918. Price, \$9.00.

This atlas of war ophthalmoscopy is a striking evidence of scientific activity and achievement under adverse conditions. The whole of the text is printed in both French and English. The diagrams serve to indicate the path of the projectile causing the lesions, and each of them is printed in connection with both the French and the English text. The primary purpose of the work is to present by means of the plates, the ophthalmoscopic appearances caused by the various war injuries. But the explanations of the exact nature of the wound, the injuries to other parts that occurred in connection therewith, the symptoms arising therefrom, and the probable character of the changes that cause these appearances, add very greatly to the interest and value of the plates.

In the first 55 pages of text, the author analyzes the leading features of these injuries for the purpose of classifying them, and bringing out certain general laws and facts relating to them. He arranges them in six groups as follows:

I. Lesions of the inner coats of the eye caused by concussion from a distance, the force reaching the eye wholly thru the air.

II. Lesions of the optic canal by injuries of the frontal region and radiating fractures of the vault of the orbit.

III. Macular and paramacular lesions, from general concussions of the bones of the face, the injury not directly involving the orbital cavity.

IV. Lesions produced by projectiles acting on the bones of the face and fracturing the orbit. These include macular and paramacular lesions thru concussion, and peripheral fundus lesions due to contact of extraocular tissues.

V. Lesions of the deeper coats by projectiles involving the orbit behind the eyeball, but without touching it.

VI. Lesions of the internal coats of the eyeball by mediate or immediate contusions of the eyeball.

With reference to each of the above groups, Lagrange deduces from his observations certain laws:

I. The commotion produced in the air by the explosion of a shell at a distance may cause laceration of the uveal tract, luxation or subluxation of the lens or traumatic cataract. The chorioretinal lesions are here emphasized because it has been disputed that they could occur from this cause. Five such injuries with hemorrhages, choroidal ruptures, and later pigmentations about the posterior pole of the eye are here represented. Macular and paramacular lesions predominate, but sometimes extend to other parts.

II. Lesions extending into the optic canal cause optic atrophy. Many of these cases have been seen, but they are generally well understood and only two are published. Other nerves in and about the orbit, both sensory and motor are often involved.

III. Eyes that appear uninjured have suffered from injuries to the bones of the face thru a transmitted vibratory concussion. Lagrange compares this to the shaking of a ship by the waves beneath it. The adipose tissue of the orbit is almost fluid at the body temperature. The predilection of such lesions for the macula is explained by the supposition that this region is the most delicate, and by the resistance to ocular displacements afforded by the optic nerve. Lesions arising in this way, but causing no changes visible to the ophthalmoscope may produce the loss of central vision. Individuals thus affected must not be regarded as malingersers.

IV. Violence, causing a thrusting in of the orbital wall, causes more extensive injuries. Along with macular lesions, which are explained as those of the preceding groups, occur lacerations of the choroid, or more often of both choroid and retina toward the periphery of the fundus, and fronting

the point of fracture in the orbital wall. These lesions may be due to actual contact of the bone with the sclera, but they are also caused by a wave movement of the intermediate soft tissues. This ruptures the choroid, which bleeds; and the blood lifts and detaches the retina, which it may in turn rupture. From such an injury there follows a proliferating chorioretinitis.

V. When the missile passes thru the orbit without touching the eyeball, it may produce lesions like those just described, and in addition do great damage to the various structures in the orbit. If it strikes the optic nerve, this is likely to be more or less completely torn loose from its scleral attachments, giving rise to the ophthalmoscopic appearances of evulsion of the optic nerve. Three of the plates show such effects.

VI. When the projectile actually touches the sclera, the most severe injuries are produced. Cases of actual destruction of the eyeball by rupture of the sclera are not here considered; but short of this both choroid and retina may be lacerated by the force of the injury. The point of contact is the center of most severe injury; and from this region lesions radiate more or less extensively to other parts of the eye. Such extensions may involve the macula; but it is not often independently affected.

The lesions studied with the ophthalmoscope are discussed under the heads: Lesions of the optic nerve; Lesions of the Choroid; Traumatic Lesions of the Retina; and Traumatic Proliferating Chorioretinitis.

The injuries of the optic nerve may occur in the intraocular portion, in the retrobulbar part containing the blood vessels, or in the part extending from where the vessels enter the nerve to the optic foramen. The evulsion of the nerve causes hemorrhage and a deep pit. But the former is absorbed and the latter fills up with proliferating connective tissue. Injuries involving the retinal vessels often give rise to the appearances of ischemia of the retina; yet this part of the nerve may be quite seriously damaged without the vessels

being much affected. With regard to hemorrhage into the optic nerve sheath, Lagrange has never observed a case in which such hemorrhage extended directly on to the optic disc, altho such cases have been recorded by other observers, his plate 23 might well represent a late stage of such a lesion. He suggests that the appearance of a pigment ring on the disc does not prove the previous extension of the hemorrhage to this part, since the pigment cells may have migrated there later.

Against the view that the choroid cannot be ruptured so long as the sclera resists the contusion, Lagrange brings forward a large experience.

More than a dozen of his plates illustrate just that accident. But in many cases in which the choroid is lacerated, the retina escapes, and in both these membranes the vessels are more likely to escape than the other tissues. The lesions produced by air concussion are generally choroidal; those of contact chorioretinal.

The clinical picture and appearances of traumatic proliferating chorioretinitis are the most valuable contribution this work makes to the literature of ophthalmology. The leading characteristics of this condition are brought out by the following comparative table:

COMPARATIVE TABLE SHOWING THE DIFFERENCE BETWEEN THE CLASSICAL PROLIFERATING RETINITIS AND TRAUMATIC PROLIFERATING RETINITIS.

Classical Proliferating Retinitis is produced by organization of the extravasated blood. It may exist without foregoing hemorrhage.

Membranes with numerous processes; appearance of a cobweb covering a large extension of the fundus.

Membranes translucent at certain points.

Protuberances ending freely in the vitreous.

Pedicles, polymorphous masses, irregular surface, marked prominence.

Is located in any part of the retina.

Foci of pigmentation rather frequent.

Is often complicated by retinal detachment produced by traction of bands.

Traumatic Proliferating Chorioretinitis is always consecutive to a hemorrhage and to a rupture of the inner membranes.

Fibrous patch of a more regular thickness.

Opaque everywhere.

Simple proximity to the vitreous.

Smooth and even surface without marked prominence.

Much more frequent in the macula, and disc and adjoining parts.

Foci of pigmentation very frequent.

Not accompanied by detachment following the proliferation; on the contrary this latter binds the retina to the choroid.

In the introduction the upper part of each page presents the French text and the lower part the English translation. The latter made by Dr. Menier, a pupil of Lagrange, shows evidence of imperfect mastery of English. Optic "canal" is rendered "optic channel" and "projectile" is translated "missile" when the use of the original words "canal" and "projectile" would better conform to the best English usage. How-

ever these defects do not seriously impair the value of the translation; and this arrangement of the two texts is very convenient for one who desires to improve his reading knowledge of French.

The explanations of the plates are printed on successive pages with the English translation facing the plate.

It is not unusual to find French works lacking in those essential con-

veniences, a table of contents and an index. This work is a glaring example of this defect. It has neither index, table of contents, nor page headings. This does not greatly matter if one sits down to read it thru from beginning to end. But if one desires to find or refer to some particular topic, it detracts greatly from the value of the work, not to have any key to what it contains.

On the whole this book is extremely valuable and welcome. The subject of ocular injuries can never be learned thru rules or generalized statements. The more cases one studies, the better fitted to deal with them he becomes, and these plates offer an opportunity to study certain things about a large series of closely related cases. E. J.

The Animal Parasites of the Human Eye, with Notes on Similar Organisms in Other Vertebrates, by **Henry B. Ward**. Large 8vo. 92 pages, with 20 illustrations. Chicago, Cleveland Press, 1918.

This book is a reprint of the article on "Parasites, Ocular," in volume XII of the American Encyclopedia of Ophthalmology, which we have already mentioned in a notice of that work (p. 455); but it is fully worthy of more extended consideration.

The number of animal parasites already known as causing ocular symptoms is large, while the probability is strong that more are to be discovered than are yet known. On the list of those of known important etiologic significance are such widely separated organisms as the malarial plasmodium, the spirochetes, the liver flukes, the filarias, the fly larvae, and the louse; and in each of the orders to which these belong are probably many others that await recognition. A systematic review of the subject by an expert biologist ought to be of the highest value in the recognition of the known organisms, and the placing in the proper class of others as they are encountered clinically. Even the organisms, the claims of which are still not established, like the trachoma bodies (chlamydozoa) are here discussed.

In the preparation of this treatise a large amount of historical material has been searched and some of its more serious errors corrected. The plate found in a sixteenth century book that was supposed to illustrate the extraction of the Guinea worm from the eye is here reproduced. It was originally published by Jean Linscot, but probably represents the custom in Ormus of putting out the eyes of possible claimants to the throne; while in another part of the picture is shown a man in the act of extracting a Guinea worm from his leg.

It is to be hoped that existence of this monograph will bring about an improvement in the literature of its subject in the direction of greater definiteness in the description of the animal parasite observed in connection with any case. If this can be identified positively with some known form that causes such symptoms this should be done with the aid of the characteristics here given. If this cannot be done or if the organism is believed to represent a new species, it should be submitted to a thoroughly qualified expert biologist. By careful treatment of the specimen this is possible from any part of the world. And any such expert will be only too glad to study a possible new claimant for recognition in the class of organisms to which he is devoting special attention. E. J.

CORRESPONDENCE.

LONDON LETTER.

The Oxford Congress.

The Oxford Ophthalmological Congress held its ninth annual meeting at Oxford on Thursday, July 11th, and Friday, July 12th, last.

As in former years members enjoyed the hospitality of Keble College, whilst the meeting proper was held in the Department of Human Anatomy kindly lent for the purpose by Professor Arthur Thomson.

The proceedings opened at 10 a. m. on Thursday with an address by the Master, Mr. Sydney Stephenson, on "The History of the Congress."

The subject chosen by the Council for the annual discussion was "Ophthalmology and the War," the breadth of the title being such as to allow of both civil as well as military points of interest being introduced.

Sir William Job Collins, K. C. V. O., M. P., who had been invited to become the Doyne Memorial Lecturer for the year, opened the discussion; and in his preliminary remarks paid a just and sympathetic tribute to the late Robert W. Doyne, founder of the Congress.

The address on the subject commanded the closest attention of the meeting and a full discussion followed in which the following took part: Lieut.-Col. R. H. Elliot, (London), Capt. E. H. E. Stack, R. A. M. C., T., Miss Marion Gilchrist, (Glasgow), Dr. T. Harrison Butler, (Leamington Spa), Capt. P. H. Adams, R. A. M. C., T., Capt. Percival J. Hay, R. A. M. C., T., Capt. Thomson Henderson, R. A. M. C., Mr. R. J. Coulter, (Newport, Mon.), Maj. A. C. Purchas, N. Z. M. C., Staff-Surg. Hanson, C. B. E., R. N. V. R., Capt. Whittington, R. A. M. C., Mr. J. Gray Clegg, (Manchester), Dr. George Young, (Colchester), and Mr. Bernard Cridland, (Wolverhampton).

At the conclusion of the discussion Sir William Collins was presented with the Doyne Memorial Medal.

A contribution by Lieut.-Col. R. H. Elliot on "Some Useful Devices in Operative and Other Work" concluded the proceedings of the first day.

On Friday, July 12th, Sir William Barrett, F. R. S., read a valuable paper accompanied by demonstrations and slides on "Entoptic Vision." Sir William Barrett's work is well known on this subject, one which has since the introduction of the ophthalmoscope been far too much neglected by ophthalmologists. But as presented by him to the Congress it is likely to be productive of valuable assistance to the practice of ophthalmology in the near future.

Col. Hanford McKee, C. M. G., C. A. M. C., followed with a paper entitled "Some Aspects of Military Ophthalmology," in which the conclusions of a wide ophthalmic experience, gained

during the war from its beginning, were given and which should be of value to the highest administrative authorities.

The morning session concluded with two papers by Capt. Percival J. Hay, R. A. M. C., on:

(a) "Implantation of cartilage after excision or evisceration of the eye."

(b) "Vulcanite casts as a support for prosthesis."

Both of these were illustrated by slides, photographs of operative results and radiograms.

In the afternoon cases were shown at the Eye Hospital by Capt. P. H. Adams, R. A. M. C., T., and Dr. Orr, after which a paper by Lieut.-Col. Elliot on "Herpes Zoster Ophthalmicus" was read, followed by another on "The Results of Cataract Operations," by Dr. T. Harrison Butler, both of which were discussed.

In the Scientific Museum, Sir William Barrett gave demonstrations with the entoptoscope and an optometer. Mr. Sydney Stephenson and Lieut.-Col. Elliot showed various microscopic sections. Capt. Thomson Henderson showed sections of monkeys' eyes with special reference to the origin of the pectinate ligament, and Capt. Adams showed sections of horses' eyes with reference to a hitherto unknown disease causing blindness.

Capt. T. Henderson showed photographs of gun-shot injuries of the eyes and orbital regions, and also of contracted sockets improved by a suture operation which he described.

Captain Stack showed a combined perimeter and scotometer together with useful novelties.

Mr. R. J. Coulter showed improved test-types.

In the Commercial Museum various novelties were on view.

The contributions to the Congress will appear in the Transactions of the Ophthalmological Society of the United Kingdom with which body the Congress is now affiliated.

The arrangements for the Congress were in the hands of the Master, Mr. Sydney Stephenson, and of the Honorary Secretary, Mr. A. Bernard Crid-

land, and were excellently carried out. About 40 members were present. The official dinner was dispensed with, on account of war conditions; but a general meeting of the members was held on Thursday evening when a number of important subjects were discussed.

R. H. ELLIOT.

OPHTHALMIC EXAMINATIONS.

The American Board for Ophthalmic Examinations at a recent meeting held in New London, Connecticut, decided to hold its next examinations at the New York Eye and Ear Infirmary, New York, Friday, October 25. Dr. William H. Wilder, Chicago, was elected secretary of the board.

The examinations next October will be the fifth to be conducted by the board. This board is composed of representatives of the American Ophthalmological Society, the Section on Ophthalmology of the American Medical Association, and the Academy of Ophthalmology and Oto-Laryngology. By arrangement with the American College of Surgeons the board has become the Ophthalmic Credentials Committee of the College, and conducts the examinations of the ophthalmic candidates for Fellowship in the College. The examinations as stated in Bulletin No. 1 of the College are as follows:

In addition to the general requirements for admission to fellowship (except Article 9), the examinations in ophthalmology consist of, first, case records; second, written examinations; and, third, clinical laboratory and oral examinations, or so much thereof as may be judged necessary:

a. Candidates in ophthalmology are required to submit twenty-five complete case records in

accordance with Article 9. Ten of these records should be of cases of ocular diseases and defects of varied character, including errors of refraction or muscle balance; external ocular diseases or diseases of the uveal tract or retina, or of the optic nerve, or glaucoma. The reports should show especially the reasons for the diagnosis and for the operative treatment, and the technique of operations.

b. The written examination will test the candidate's knowledge of the underlying principles or science of ophthalmology, including anatomy, embryology, physiology, physiologic optics, pathology, relation of the eye to other organs and diseases of the body.

c. The oral examination will include:

The external examination of the eye.

Ophthalmoscopy: (Candidates are requested to bring their own ophthalmoscopes).

Measurements of errors of refraction.

Testing of the ocular movements and fields of vision.

Relations of ocular conditions to diseases of other parts of the body and their treatment.

Laboratory examination in histology, pathology, and bacteriology of the eye."

Further information may be had upon request from the American College of Surgeons, 25 East Washington Street, Chicago.

TO FIX ROUND LENSES.

To the Editor:

Since writing the article for the July number of the JOURNAL about preventing the turning of round lenses it has been discovered that the drop of solder can be used in the Windsor frame either by removing the celluloid rim or without doing so, if the solder be applied by means of a copper wire used like a soldering iron.

Yours very truly,

DAVID W. WELLS.

Boston, Mass.

NEWS ITEMS

Personals and items of interest should sent to Dr. Melville Black, 424 Metropolitan Building Denver, Colorado. As these columns go to press on the 30th of the month contributors should send in their items by the 25th. The following gentlemen have consented to supply the News Item Editor with the news from their respective sections: Dr. H. A. Beaudoux, St. Paul; Dr. V. A. Chapman, Milwaukee; Dr. A. E. Davis, New York City; Dr. Robert Fagin, Memphis; Dr. M. Feingold, New Orleans; Dr. Wm. F. Hardy, St. Louis; Dr. George F. Keiper, La Fayette, Indiana; Dr. George H. Kress, Los Angeles; Dr. W. Holbrook Lowell, Boston; Dr. G. Oram Ring, Philadelphia; Dr. Chas. P. Small, Chicago; Dr. George M. Waldeck, Detroit. It is desirable that this staff shall be enlarged until every city of importance in the United States shall be covered, as well as foreign countries. Volunteers are therefore needed and it is hoped that they will respond promptly to this call.

DEATHS.

Professor Stephan Bernheimer, of Innsbruck, died recently.

Colonel Raymond C. Bolling died while on duty with our Aviation forces in France.

Arthur W. De Roaldes, New Orleans, aged 69, died at his home, June 13th.

Charles H. Williams, Boston, aged 67, died at his home in Cambridge, June 9th, from heart disease.

Edwin G. Cowperthwaite, Philadelphia, aged 44, died at his home, June 11th.

James J. Smith, of New Britain, Conn., aged 55, died at the Howard Hospital, May 23rd, from cerebral hemorrhage.

Dr. Francis Barraza, Adjunct Professor of Ophthalmology in Buenos Aires, is dead. His principal papers were: "Ocular Trauma Under the Medico-Legal Aspects," and "Etiology of Myopia." He devoted also a great deal of his time to chemistry.

Dr. J. Gonzales Castellanos of Valencia, Spain, passed away at the age of 85 years. He was a pioneer in fighting trachoma since 1850. In 1870 he founded a free clinical course of Ophthalmology in Valencia, the first in Spain of its kind. Afterwards he devoted himself to the study and treatment of lepers.

PERSONAL.

Dr. J. W. Newman was recently reelected President of the Louisiana Commission for the Blind.

Dr. F. M. Fernandez has been awarded a prize of the Academy of Sciences of Havana for his essay "On the Etiology and Treatment of Strabismus."

Jos. D. Heitger, A. B., M. D., announces the removal of his offices from Bedford, Indiana, to the Atherton Bldg., 608 Fourth street, Louisville, Ky.

Dr. Cassius D. Wescott, of Chicago, who recently was obliged to undergo an opera-

tion, is recuperating rapidly, and expects to resume his practice in August.

Dr. A. H. Little, Portland, Maine, has received his commission as Captain, M. R. C., and has gone on active duty at Camp Devens, Massachusetts.

Dr. J. A. Spaulding, of Portland, carried thru to a most successful ending as President, the annual meeting of the Maine Medical Association in June, besides contributing two important papers for discussion. His annual address, "The Eyes in War," will appear in a subsequent number of this Journal.

Drs. H. H. Briggs, of Asheville, N. C., F. P. Calhoun, of Atlanta, Ga.; Dorland Smith, Bridgeport, Conn.; David N. Dennis, Erie, Pa.; D. F. Harbridge, Phoenix, Arizona; J. W. Jervy, Greenville, S. C.; Wm. F. Hardy, St. Louis, Mo.; W. E. Shahan, St. Louis, Mo., and Hunter H. McGuire, of Winchester, Va., have been elected to membership in the American Ophthalmological Society.

Dr. S. J. Beach, Augusta, Maine, is doing valuable work on the newly formed State Board of Health and in conjunction with the Association Committee on Conservation of Vision. Dr. Spaulding, of Portland, intends to introduce that invaluable topic into the Normal and High Schools of Maine, Educating the Teachers and Scholars by Means of Lantern Slides." It is interesting to note that this is the first instance so far advanced in which a Board of Health has gone into the propaganda of Conservation of Vision.

SOCIETY NEWS.

A new Ophthalmological Society has been founded in Madrid. The proceedings are published in the April number of "Archivos de Oftalmologia Hispano-Americanos."

Because of the fact that more than half of the membership of the Sioux Valley Eye and Ear Academy are in active army service, there was no meeting of the Academy at Omaha in July.

Before the Section on Ophthalmology of the Canadian Medical Association at its recent meeting in Hamilton, Ontario, May 29th to 31st, papers were read by Col. W. R. Parker, of Detroit, on "Treatment of Simple Glaucoma," which will appear later in the *AMERICAN JOURNAL OF OPHTHALMOLOGY*; by Dr. E. E. Blaauw, of Buffalo, on "A Rare Growth on the Cornea," presenting patient and microscopic slides, and by Dr. John Wheeler, of New York, on "Paralysis of Accommodation."

MILITARY NOTES.

Major George F. Suker, of Chicago, has been transferred from Camp Custer to Hoboken, N. J.

Captain Chas. W. Kollock, of Charleston, S. C., is in charge of the Physical Examining Unit for the Aviation Section of the Signal Corps at Charleston.

Additions to the Honor Roll from Indiana Ophthalmologists:

Charles J. Adams, Washington,
C. N. Howard, Kokomo,
J. W. Green, Warsaw,
R. E. Swope, Rockville,
Harry Boyd Snee, South Bend,
A. E. Barber, South Bend,
W. C. Dyer, Evansville,
T. P. Goran, Richmond.

Dr. Sidney Walker, Jr., of Chicago, who was seriously wounded during a recent engagement on the western battle front, is at present in a hospital in the outskirts of Paris. He is reported as out of danger and able to get around.

Dr. James A. Smith, of Chicago, who received a Lieutenant's commission in the Medical Reserve Corps in June, has been ordered to report for duty at Fort Oglethorpe.

Dr. Henry C. Haden, of Galveston, Texas, is now in service at the Walter Reed General Hospital, Washington, D. C., with rank of Major.

The names of Lieut. C. A. Bahn, of New Orleans; Capt. D. C. Iles, of Lake Charles, La.; Capt. A. H. Little, of Portland, Me.; Capt. George E. Frothingham, of Detroit, should be added to the Honor List of Ophthalmologists now serving in the U. S. Army.

MISCELLANEOUS

Col. James Bordley at a recent meeting held at the U. S. Army General Hospital No. 7 at Evergreen, outlined a plan of far-reaching scope which will make Baltimore the center for the reeducation of all war blinded soldiers.

The new health law of Kentucky, recently enacted, included inspection of the eyes of school children among the duties to be imposed upon the county or district health officer who shall be appointed under that law.

The Eleventh Annual Report of the Massachusetts Commission for the Blind is full of interest. "For five years the Commission has been making studies in various cities of Massachusetts among children with seriously defective sight, and has been urging the creation of special classes in the public schools where the number of such handicapped boys and girls makes it possible. Three such classes are now in existence in Boston, one in Cambridge, one in Lynn and one in New Bedford. These classes have a total membership of 78." Thus far the Commission has dealt with 919 children with seriously defective vision. Every effort should be made to include all cities to establish one or more sight saving classes.

OPHTHALMIC LITERATURE

Under this head continuing the "Index of Ophthalmology" heretofore published in **Ophthalmic Literature** will be found the subjects of all published papers received during the last month, that bear to an important extent upon ophthalmology. The subject is indicated rather than the exact title given by the author. Where the original title has been in a foreign language it is translated into English. The journal in which the paper is published will indicate the language of the original.

The names of the different journals are indicated by abbreviations which generally correspond to those used by the **Index Medicus**, the **Journal of the American Medical Association**, and the **British Journal of Ophthalmology**. We will from time to time publish the list of ophthalmic journals, with the abbreviations used for each. Often a single letter discriminates between journals published in different languages. Thus "Arch. of Ophth." refers to the Archives of Ophthalmology, published in English; "Arch. d'Ophth." indicates the French Archives d'Ophthalmologie; "Arch. de Oftal." refers to the Archivos de Oftalmologia Hispano-Americanos, while "Arch. di Ottal." indicates the Italian Archivio di Ottalmologia.

In this index of the literature the different subjects are grouped under appropriate heads; so that all papers bearing on the same, or closely related subjects, will be found in one group. The succession of the groups is the same from month to month, and identical with that of the Digest of the Literature. Where a paper clearly refers to two subjects that belong in different groups, it will be noticed in both groups.

Each reference begins with the name of the author in heavyface type. This is followed by the subject of his paper. Then in brackets a number with (ill.) indicates the number of illustrations, or a number with (pl.) the number of plates illustrating the article, (col. pl.) indicates colored plates. (Abst.) shows that it is an abstract of the original article. (Bibl.) tells that the paper is accompanied by an important bibliography. (Dis.) means that the paper was read before some society and gave rise to a discussion which is published with it.

The "repeated titles" may render accessible the essential part of a paper, the original of which could not be consulted. These give (in brackets) after the author's name the volume and page of this department of "Ophthalmic Literature" where the title of the paper will be found; and then the journal, volume, and page where the translation or abstract is published.

It is desired to notice every paper as soon as possible after it is published. Readers will confer a favor by sending titles they notice have been omitted, with journal and page of publication; and of their own papers, sending either a copy of the journal in which each appeared, or a reprint. These should be sent as soon as possible to 318 Majestic Building, Denver, Colorado.

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ABBREVIATIONS

The following are the abbreviations used in Ophthalmic Literature for the names of the journals most frequently referred to. The names of the principal ophthalmic journals now published are here included, with those of journals merged to form this journal; and of a few general medical journals which will illustrate the general plan of choosing such abbreviations. The same plan is followed when journal names are abbreviated in the bibliographies appended to original papers.

This list also indicates, in most instances, how often the journal is issued and the city of its publication, facts not given in the abbreviations. When any such abbreviation is used the volume and page are appended, thus v. 1 p. 387, or in a few cases the date of publication. It is manifestly impossible to give here abbreviations for all the many hundreds of general medical journals. The few appended must be regarded as samples of such abbreviations. It will be the endeavor of our compilers always to give enough of the journal name to

make it possible to identify the journal with certainty in the catalogue of any library, or any dealer through whom it might be obtained.

- Amer. Jour. Ophth.** American Journal of Ophthalmology. Monthly, Chicago. In general this journal (series 3) is referred to. When, however, the number of the volume is higher than that of the current volume, the reference is to its predecessor, published in St. Louis. When there would otherwise be uncertainty, or if the first journal of this name is referred to, the year of publication will be appended.
- Ann. of Ophth.** Annals of Ophthalmology. Quarterly, St. Louis.
- Anales de Oftal.** Anales de Oftalmologia. Monthly, Mexico.
- Ann. d'Oculist.** Annales d'oculistique. Monthly, Paris.
- Ann. di Ottal. e Clin Oculist.** Annali di Ottalmologia e Clinica Oculistica. Monthly, Rome.
- Arch. d'Ophthal.** Archives d'ophthalmologie. Monthly, Paris.
- Arch. of Ophth.** Archives of Ophthalmology. Bi-monthly, New York City.
- Arch. f. Augenh.** Archiv für Augenheilkunde. Irregular, Wiesbaden.
- Arch. di Ottal.** Archivio di Ottalmologia. Irregular, Palermo.
- Arch. de Oftal. Hisp.-Amer.** Archivos de Oftalmologia Hispano-Americanos. Monthly, Barcelona.
- Brit. Jour. Ophth.** British Journal of Ophthalmology. Monthly, London.
- Clin. Oculist.** La Clinique Oculistique. Monthly, Paris.
- Centralb. f. prakt. Augenh.** Centralblatt für praktische Augenheilkunde. Monthly, Leipzig.
- Graefe's Arch. f. Ophth.** Archiv für Ophthalmologie. (Established by Albrecht von Graefe.) Irregular, Wiesbaden.
- Klin. M. f. Augenh.** Klinische Monatsblätter für Augenheilkunde. Monthly.
- Nippon Gank. Zasshi.** The Journal of Japanese Ophthalmologists. Monthly, Tokyo.
- O. L.** Ophthalmic Literature. Monthly, Denver.
- Ophth. Rec.** Ophthalmic Record. Monthly, Chicago.
- Ophthalmol.** Ophthalmology. Quarterly, Seattle.
- O. Y. B. or Y. B.** Ophthalmic Year Book. Annual, Denver.
- Soc. Buenos Aires.** Boletín de la Sociedad de Oftalmologia de Buenos Aires. Annual. Buenos Aires.
- Soc. franc. d' Opht.** Bulletins et mémoires de la Société française d'ophthalmologie. Annual, Paris.
- Trans. Amer. Ophth. Soc.** Transactions of the American Ophthalmological Society. Annual, Philadelphia.
- Trans. Ophth. Soc. U. K.** Transactions Ophthalmological Society of the United Kingdom. Annual, London.
- Vestnik Ophthalmol.** Vestnik Oftalmologii. Bi-monthly, Kiev.
- Woch. f. Therap. u. Hyg. d. Aug.** Wochenschrift für Therapie und Hygiene des Auges. Bi-weekly. Breslau.

GENERAL MEDICAL JOURNALS

- Amer. Jour. Med. Sc.** American Journal of the Medical Sciences. Monthly, Philadelphia.
- Boston Med. and Surg. Jour.** Boston Medical and Surgical Journal. Weekly, Boston.
- Brit. Med. Jour.** British Medical Journal. Weekly, London.
- Jour. A. M. A.** Journal of the American Medical Association. Weekly, Chicago.
- Lancet.** The Lancet. Weekly, London.
- Nederl. Tydschr. v. Geneesk.** Nederlandsch Tydschrift voor Geneeskunde. Weekly, Amsterdam.